# Brain

## A Journal of Neurology

CONTENTS


**Phantom Limbs and Body Shape.** By GEORGE RIDDOCH .. .. .. 197

**Cerebral Electrical Changes in Experimental Concussion.** By DENIS WILLIAMS and D. DENNY-BROWN.. .. .. .. .. .. .. .. 223

**The Component Reflexes of Micturition in the Cat.** Part III. By F. J. F. BARRINGTON .. .. .. .. .. .. .. .. .. 239

**Visual Disorientation with Special Reference to Lesions of the Right Cerebral Hemisphere.** By W. RUSSELL BRAIN.. .. .. .. .. .. 244

**Akinetic Mutism with an Epidermoid Cyst of the 3rd Ventricle.** By H. CAIRNS, R. C. OLDFIELD, J. B. PENNYBACKER and D. WHITTERIDGE.. .. .. 273

**A Note on the Clinical Anatomy of the Veins, with Special Reference to the Spinal Veins.** By H. A. HARRIS .. .. .. .. .. .. 291

**Index** .. .. .. .. .. .. .. .. .. .. .. .. 301



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ASSISTANT EDITOR

J. G. GREENFIELD, M.D.

WITH THE HELP OF

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SIR J. PURVES-STEWART, M.D.
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All Editorial Communications, Manuscripts and Printed Matter to be addressed to the *Editor*, **F. M. R. WALSHE, M.D., 11-12, Wimpole Street, W.1.**

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DECEMBER, 1941.

# BRAIN

VOL. 64, PART 4.

## PHANTOM LIMBS AND BODY SHAPE.[1]

BY GEORGE RIDDOCH.

### INTRODUCTION.

KNOWLEDGE of the persistence, as a phantom, of a limb which has been removed must be as old as survival from amputation. Nevertheless, it is an interesting and, at first, surprising fact that medical literature up to comparatively recent times has been almost silent on the subject. The phenomenon, it is true, must have been mysterious to our forefathers, whose physiological and psychological advantages were less than our own. But the mystery could not have lain in the purely subjective nature of the symptoms alone, for other subjective sensations and experiences were often the basis of penetrating inquiry. What must have constituted the apparently preternatural character of a phantom was the seeming persistence of a part that was known to be gone, and the continued possession of many of its original "attributes" of form, position, and even voluntary movement; all the more when it was the seat of excruciating pain. Such a state of affairs was beyond reason and it would not be surprising if the unfortunate patient was regarded as an obstinate, lying fellow or even possessed of the devil. In fact, it was a matter that was better left alone.

The patient himself must have often distrusted the reality of his own sensations, and even to-day amongst the uneducated classes spontaneous description of phantoms is rarely offered unless they are painful. Dread of the unusual, of disbelief, or even of the accusation of insanity may be behind this reticence, and the relief which follows free discussion of the patient's symptoms, as natural experiences, must be a commonplace to those who are interested in the subject.[2]

[1] Being the Presidential Address, Neurological Section, Royal Society of Medicine, 1941.

[2] Admiral Lord Nelson believed that the phantom fingers of his amputated arm provided "a direct proof of the existence of the soul."

This guarded attitude of the patient is comprehensible, but what is surprising, in these days of advanced physiological knowledge, is the frequent refusal of medical men to believe that phantom limbs are anything more than psychological abnormalities. Phantom-illusions, it is true, are not uncommon symptoms in insanity and hysteria and have been studied by psychiatrists; but they belong to a clinical group separate from that which includes the phenomena about to be described. They are related only in so far as they are both disorders of the recognition of body shape. The phantom-illusion of the psychotic, however, is the outcome of gross mental disturbance, whereas the phantom which follows a lesion of sensory paths, with or without amputation of the limb, is determined essentially by upset of physiological processes and its explanation must be primarily searched for on that basis.

## Clinical Description of Phantom Parts of the Body following Lesions of the Nervous System.

In approaching this subject from a clinical point of view, it would be well first to consider the varieties of phantoms which are best known—namely, those which follow amputation of limbs. Whether amputation has been the result of an accident or operation, it is usual for the patient, at least for a time, to experience sensations as if the limb were still present. These sensations may be either painless or painful, but are always more or less abnormal.

(*a*) *Painless phantom limbs after amputation.*—Let me describe an illustrative case. A man, aged 48, a Methodist parson of somewhat rigid and unimaginative outlook, had his right leg amputated at the junction of the middle and lower thirds at the age of 14. From then onwards (and I examined him thirty-four years later) he had been aware of a phantom right foot, the hallux and instep being most clear. The remaining part of the amputated limb segment, namely the lower part of the leg and the ankle, were not represented. The phantom closely resembled in many ways his real foot. It was correctly placed in relation to his stump with which it moved, and, on voluntary effort, he could flex and extend the phantom foot and toes. When he was particularly fit or pleasantly excited, his phantom foot might tingle like the rest of his body, and when his mind was fully occupied he was as unaware of his phantom as he was of his real body. A year after the amputation he was fitted with an artificial limb and the phantom then seemed to coincide with the artificial foot. When wearing the artificial limb, he was able to differentiate, by treading on them, between objects of grossly varied shape, such as a

button or match, placed on the floor. He was accustomed to remove the appliance in the evening and to sit with his right amputated leg crossed over his left. If he had occasion to get up suddenly from his chair, he often stepped off with his phantom foot, so natural did it appear to him. In bed it used to itch more than his left foot and involuntarily he would put down his hand to scratch it. Nevertheless, the foot was never quite normal. It seemed to be broader than the other and, under certain circumstances, was the seat of uncomfortable sensations. Thus, before a spell of frost his toes felt crushed as if by a tight shoe. He also experienced a sensation of coldness in the outer edge of his foot, such as might occur if the boot were split. When the frost became well established, these sensations disappeared but, in a long frost, they might persist for several days. They temporarily diminished or disappeared if he toasted his leg in front of the fire. Again, before rain he had the sensation as if his foot and toes were incompletely immersed in water which was being gently whirled round. Windy weather evoked the same sensations as frost, except that his phantom toes then felt separated from one another. All these abnormal sensations were more obtrusive in the winter, and so accurate were they that he had gained a local reputation as a weather prophet. He also claimed to be able to foretell any change in the weather by a tendency for his toes to curl down.

In regard to the stump, there was no discoloration, marked wasting, twitching or spontaneous pain. A bulb on the sciatic nerve about an inch above the end of the stump could, however, be felt. Pressure on it at first gave rise to a sensation of numbness in the whole phantom foot, and, if continued, to a more localized sensation. Thus, if the pressure were on the inner, middle or outer aspects of the bulb, the sensations became more prominent in the little toe, middle toes and hallux respectively.

This case is a good example of a painless phantom limb after amputation except that it was of unusually long duration. As a rule, a painless phantom after a few weeks gradually becomes less noticeable and, at the same time, approaches the stump into which it finally disappears and fades away. If, however, soon after this happens the patient's health becomes impaired, for example, from influenza, fatigue or worry, the phantom part may temporarily reappear. Stimulation of the stump by pressure on the nerves or faradization of them, as Weir Mitchell first pointed out, has the same effect, and if the phantom is still appreciated, but has approached the stump, it will then temporarily lengthen, to shorten again when stimulation has ceased [4]. Further, the wearing of an artificial limb which is comfortable will often, but not invariably, cause a shortened

phantom to regain its correct distance from the stump and to become exactly incorporated in the artificial limb.

A phantom part, after amputation of a limb, is the rule, but there are exceptions, more frequent perhaps with high than with low amputations, and especially with disarticulations at shoulder or hip. According to Lhermitte and Susie [7], phantoms are sometimes obvious at night only, perhaps, as in the case of mild tinnitus, because attention is then less diverted; or, again, they may be noticed only when paræsthesiæ are referred to them.

The patient is aware of his painless phantom immediately or soon after his limb has been removed. Sometimes it consists of the whole absent part, but much more often of the peripheral portion alone. In some cases the hand or foot is incomplete, and then the thumb-index, hallux-heel segments tend to remain. Although the patient is not aware of the forearm or leg, the hand or foot is commonly described as being, for a time at least, correctly placed in relation to the stump with which it moves in unison. Occasionally, from the first it is described as being situated nearer to the stump than it ought to be, or even to be within the stump, but this is exceptional at this stage. Solid objects form no obstacle to it, so that in cases of amputation above the elbow if the phantom hand appears to be in front of the body, as if the elbow were flexed, and the stump is moved backwards, the hand seems to pass through the trunk without any sense of obstruction. So also it is not affected by placing a hand over the end of the stump. Equally striking is the fact that the patient usually has the illusion of being able voluntarily to move his phantom, but the actions which appear to be carried out are always less complex and varied than normal. Thus, he may be able to flex and extend his fingers and thumb, or foot and toes, but finer adjustments or movements of separate digits, except of the thumb, index or hallux, are usually impossible. Gross rather than delicate movements are preserved, although occasional exceptions to this rule are met with. Associated movements may also be described, especially in relation to the paired limb. Thus, in the case of a phantom hand, strong flexion against resistance of the sound arm may lead not only to abduction of the stump and the phantom with it, but also to correct synergic movements of the phantom fingers. Contrariwise, as in Charcot's case [3], flexion of the phantom hand may evoke a corresponding associated movement of the real hand. Involuntary spontaneous movements, however, are in my experience rare, but are less so with painful phantom limbs.

In general, painless phantoms are said to feel more or less normal.

Thus, in the case just quoted, the patient was apt to forget that his foot was not real and attempted to use it in the ordinary way; and another man with a phantom hand often unconsciously brought it to the aid of his other hand if it were in difficulties. The phantom may even seem to sweat when the body perspires, and its temperature may appear to alter with that of the stump, although this is by no means the rule. But abnormalities are always present, both sensory and motor, in the form of paræsthesiæ, alterations in size, in relation to the stump, limitation of movement, and so on. A very common quasi-normal sensation is that of itchiness of the sole of the hand or foot when the patient becomes warm in bed, and he may unwittingly attempt to scratch his phantom hand or foot; but the itchiness of the phantom is abnormal in degree and frequency as compared with that in the real hand or foot. Changes in the weather, ill-health, fatigue or anxiety, in fact, any moderate alteration in the patient's physical or mental well-being, are often accompanied by additional abnormal and sometimes uncomfortable sensations in the phantom part. It may then be said to be swollen or compressed, or it may appear to tingle or twitch.

Bilateral amputation may be followed by bilateral phantom hands or feet; or one or both may be absent; or one may be painless and the other painful, the latter usually associated with tender abnormalities in the stump.

(*b*) *Painful phantom limbs after amputation.*—In nearly half the cases after amputation, whether traumatic or surgical, pain is referred to the phantom part, and the proportion is greater with upper than with lower limbs.

The pain appears contemporaneously with the phantom, immediately or soon after operation, and is complained of mainly in the hand or foot; but in contrast to the case of painless phantoms, the whole absent limb may be represented and be more or less the seat of pain. In the hand, pain is referred chiefly to the wrist, palm, knuckles and tips of the fingers and thumb, and in the foot, to the instep, heel and toes. If there had been a painful wound in the forearm or leg before the amputation, the pain may seem to persist in the same area in the phantom as if the sore were still present. The continuation of preamputation pain in any part of the limb is indeed the general rule, and, in fact, with regard to both pain and the prevailing posture of the part, it is as if the limb had not been removed.

In all but the slighter cases the pain is continuous, but subject to exacerbations. The continuous pain, which forms the painful back-

ground, is commonly described as a dull ache, or, more often, as burning and throbbing, and, frequently, the hand or foot seems to be the seat of painful rigidity or cramp, which may wax and wane. Thus, the thumb and fingers may be said to be tightly flexed with the nails cutting into the palm, and the patient may believe that he would get relief if his fingers would only relax. At the same time, if he attempts to move his phantom fingers the pain increases. Livingston [8] discovered that this distressing rigidity in a phantom upper limb could be diminished, or even abolished for a time, by injection of the upper thoracic sympathetic ganglia with 1 or 2 per cent. procaine hydrochloride, which also produced a warm sensation in the stump and phantom with diminution or abolition of pain in both. Sometimes the phantom part feels swollen and tense as if the hot, dry skin were about to burst open, or the hand or foot may seem to be bruised and crushed, as if held in a vice. The skin, instead of being dry and hot, may sometimes feel moist with sweat, and a patient may say that his phantom part perspires in tune with the rest of his body. In about one-third of Weir Mitchell's [10] cases, cooling or warming of the stump was accompanied by a similar alteration of thermal sensation in the absent part. More commonly, however, the temperature of the phantom does not appear to conform to that of the body or even of the stump.

The continuous pain varies in severity and, in addition, periodic cutting or darting pains aggravate the patient's distress, or he may feel as if his absent fingers or toes are being torn off. These paroxysms may occur for no apparent reason, but usually they are evoked by obvious stimulation. Thus, palpation of the stump, which is often red and tender and contains sensitive nerve end-bulbs or adherent scars, warming or, less often, cooling the stump or even allowing it to hang down for long, are potent exciting causes of additional pain. Sometimes such actions as yawning, micturition or defæcation will temporarily accentuate the pain, perhaps through associated muscular contraction in the stump or vascular congestion from venous obstruction. Equally important, as aggravating causes, are fatigue, sleeplessness or persistent worry, and even apprehension from the knowledge that the stump is about to be examined. In consequence, the unfortunate sufferer is not only a prey to the ordinary disturbances of everyday life, but is continually fearful of their occurrence. Emotional instability, in varying degree, is therefore an inevitable complication, as in all disorders in which incessant severe pain is a prominent symptom. From this description, it must be clear how closely the pain and associated phenomena, which are referred to the phantom, resemble those of causalgia from lesions of the median and internal popliteal nerves.

The posture of the phantom part is almost invariably that of the limb before amputation, especially if pain was antecedent to operation. Thus, a soldier whose right arm was blown off by the premature explosion of a bomb, which he had been holding in his hand, felt as if his painful phantom hand were still grasping the bomb and he could not alter its position. So also, in the case of a hospital nurse, although her left arm was amputated three years after the onset of the original local infection, during which time she had continuous severe pain in her hand, removal of the arm failed to alter either the posture of her hand and fingers, or the pain which she referred to them. Thus, if the absent part is the seat of pain irrespective of whether the amputation was immediate or delayed, the hand or foot remains "frozen" in the position it held immediately before amputation. In general, with high amputations the painful phantom upper limb is flexed and the lower limb extended, probably because of the prevailing posture before operation. Further, in contrast to the preservation of more or less free movement in painless phantoms, the presence of pain renders voluntary movement almost, if not completely, impossible. Sometimes, limited displacement of the fingers or toes can be carried out, but invariably such attempts aggravate the pain. It can be said that fixation of posture and limitation of voluntary movement of the phantom is proportionate to the degree of continuous pain and still more to that of painful rigidity. If, however, the pain is successfully abolished by lateral chordotomy or early excision of tender neuromas in the stump, free movement of the painless phantom is usually possible. Another striking consequence of pain is that there is less tendency, as time goes on, for the absent limb to shorten. Sometimes, it is true, a painful phantom hand or foot may from the time of operation appear to be nearer to the stump than it ought to be, a phenomenon which seems to be related to high amputation and to pain of, at most, moderate severity.

When a painful phantom limb is nearer to the body than it ought to be, in other words, when it is abnormally short, irrespective of whether it is in apparent direct contact with the stump or is separated from it by a gap, accentuation of pain in it is often associated with temporary elongation to the natural position. The wearing of an artificial limb may also lead to the same result as occurs, more frequently, with painless phantoms. This, however, is exceptional to the general rule of which the following is an example. A nurse, who had had a painful phantom foot for some years after amputation through the upper third of her thigh,

and wore an artificial limb, never felt that her foot coincided with the artificial foot, but that it was placed about two inches above it.

Nevertheless, in contrast to what is common in painless phantoms, when pain is referred to the absent part, and especially if it is severe and continuous, the hand or foot tend to remain correctly placed in regard to distance from the stump, and more proximal parts of the phantom limbs, if they too are the seat of pain or discomfort, are also represented. In other words, the presence of painful or uncomfortable sensations leads to persistence of the phantom, retention of the correct length of the limb, and, lastly, limitation of apparent movement of it from voluntary effort. On the other hand, involuntary movements are more common in painful phantom hands and feet, as well as of the stump, whilst linkage of the phantom with the stump in voluntary or associated movement is the same, whether there is pain or not.

### The Phenomena of the Stump.

The condition, after amputation, of the remaining portion of the limb, especially of its peripheral part, has in some respects an important bearing upon the phantom limb. In the first place, it is commonly believed that the length of the stump will determine, to some extent, whether or not a phantom part will follow operation. Phantoms are said to be more constant with low than with high amputations. In my experience, however, this statement applies more to painless than to painful phantoms. When pain has been present in the injured part, especially in the hand or foot, for some time prior to amputation, then a painful phantom can almost certainly be predicted, whether the remaining stump is short or long.

Certain features of the stump, which may appear sooner or later after the operation, influence the late development of pain in the absent part. That is to say, there may be at first a painless phantom which, within a few weeks, becomes painful coincidental with the alterations in the stump about to be described; or the painless phantom may have, for a time, disappeared only to reappear later with the development of pain.

End-bulbs or neuromas are invariably present in amputation stumps and can usually be palpated, especially when the stump is thin from wasting of the soft tissues. They consist of a mass of fibrous tissue with fine, wavy neurofibrils and must be looked upon as the normal method of healing of the proximal cut end of a nerve. They may be small and benign or abnormally large and tender. According to Trotter [16], this pathological change is a manifestation of the inherent antagonism between

neuroblastic and mesoblastic tissues, and is due to non-insulation of neurofibrils from the absence of the nerve sheath. Tender neuromas are usually associated with painful phantom limbs. Adherence of end-bulbs to the scar of the amputation wound is another source of pain from traction in movement of the stump which, in consequence, tends to be kept immobile and for this reason alone wastes and becomes blue and cold.

When prolonged sepsis is a complication, before or after amputation or both, a train of events is set agoing which greatly increases the liability to pain in the stump and the phantom. Not only the end-bulbs, but the nerve trunks become enlarged and exquisitely tender, the enlargement spreading upwards from the end of the stump. "Spontaneous" pain is added to tenderness from the stimulation of interstitial neuritis. The overlying skin is red and over-sensitive so that even gentle contact may be intolerable. The pain is increased by congestion from heat or dependence of the mutilated limb, so that the patient prefers to keep it uncovered and elevated, especially in bed. Whilst gentle cooling gives relief, cold also accentuates the pain and patients frequently complain of the harmful effect of east winds, a fact which Weir Mitchell [10] recorded. It is when the stump is in this state of hypersensitiveness from neuritis that involuntary movements of it are particularly apt to occur. They vary from fascicular twitchings to coarse jumps and irregular jerkings which may be intermittent or continuous and are made worse by sudden emotional disturbances, palpation of the stump; or, in fact, any stimulus which aggravates the pain in the limb and phantom part.

The worst cases of painful phantom limbs tend to be associated with these abnormalities in the stump; and, as has already been mentioned, any stimulus, physical or emotional, that can evoke pain is usually effective for both stump and phantom limb. They are bound together in painful harmony. It is then that cramp-like rigidity of the phantom hand and fingers most often forms the constant background of pain. There are rare cases, it is true, of tender stumps, usually of the lower limb, in which phantoms are absent or at most periodic, but the tenderness may then be due to abnormal end-bulbs alone without the additional complication of interstitial neuritis. Sometimes also the reverse is found, namely, painful phantoms with apparently normal stumps. In this category, amputation of upper limbs provides most examples, and a constitutional and often inherited defective resistance to pain and discomfort can usually be traced in the past history.

### The Effects of Proximal Lesions of Sensory Paths on Phantom Limbs.

Because of the bearing of anæsthetization of the stump on pain in phantom limbs, it would be convenient at this stage briefly to consider the effects of division of sensory paths at proximal levels on post-amputation phantom limbs, whether painless or painful.

When the only abnormalities of a stump are tender end-bulbs or adherent scars, early excision of them may remove the pain from a phantom which then remains, for a time at least, as a painless phenomenon and may subsequently disappear in the usual way. If, however, there is interstitial neuritis, especially of long standing, no benefit will be gained by even extensive division of peripheral nerves, plexus or spinal roots, even when the anterior, as well as the posterior, roots are dealt with. As a rule, pain in the phantom part then persists unabated in spite of almost complete anæsthetization of the stump.

A frequent and startling result of destruction of roots or plexus sufficient to give rise to extensive loss of all forms of sensation with complete loss of postural sensibility in the stump, is detachment of the phantom from the stump in voluntary or passive movement of the latter. Its position in relation to the body, but rarely its shape, may also become changed and the patient may find that he can no longer move his phantom fingers. Should postural sensibility later return in the stump, the phantom again comes to move with it. That dissociation of the phantom from the remaining portion of the limb is dependent upon complete loss of postural sensibility in the shoulder or hip joints is demonstrated in cases of tabes in which this is found with resulting gross ataxia but relative preservation of other aspects of sensation.

Section of the contralateral spinothalamic tract gives the opposite effect by rendering the phantom painless without separating it from the stump. It not only still moves freely with the stump and retains its original position in relation to it, but also, like other painless phantoms, can now be voluntarily moved by the patient. For this result to be achieved, lateral chordotomy must be sufficiently high to give rise to complete analgesia not only of the stump itself but also of the neighbouring part of the trunk.

So far lesions of the peripheral and central nervous systems have been shown to lead to important alterations in phantoms which, however, have still persisted. Immediate disappearance of a phantom limb, at all events a painless one, can, so far as is known, only follow a severe and extensive lesion of the sensory cortex of the opposite hemisphere. Illustrative

examples are of necessity very rare, but Head and Holmes' [5] striking case can be quoted. A man who had a painless phantom left foot following amputation of his leg, some time later sustained extensive injury to his right parietal cortex, with consequent loss of postural sensibility in his left upper and lower limbs, and from that moment his phantom foot disappeared.

### Phantoms After Amputation of Other Parts.

Parts of the body, other than the limbs, may after removal appear to remain, as phantoms, a fact which was known to Weir Mitchell [10].

As in the case of the extremities, the lost organs continue, as a rule, to be represented, if the phantoms are painless, in their peripheral portions only, that is to say, in those parts which are most heavily endowed with sensory end-organs. Thus, after amputation of the breast the nipple alone remains, of the penis the glans, and of the nose its nostrils and tip. Commonly such phantoms, for so long as they persist, do not appear to alter in position, but exceptions are occasionally met with. For example, Weir Mitchell [10] mentions a case which had been described to him by a colleague in the American Civil War of a phantom penis which sometimes became erected, presumably with turgescence of the stump. Uncomfortable sensations may also be described, as in the case of a woman with a phantom nose who attended my clinic at the London Hospital. Her nose had been destroyed by lupus, and, for years, she had worn an artificial one which was so comfortable that she had become thoroughly accustomed to it and was, as a rule, unaware of its presence. At times, however, it felt itchy round the nostrils and unconsciously she was apt to put up a finger to scratch it. But, so far as I know, pain is not a feature of these non-limb phantoms, except in regard to teeth, which persist in this guise only when painful.

It will be noticed that the portions of the body which tend to " remain " after amputation are those which protrude or stand out from the general mass of the trunk and head. As will be seen, this fact has a bearing upon the interpretation of these phenomena.

### Phantom Parts Without Amputation.

A comparatively unexplored field is the investigation of phantoms of limbs or segments of limbs which have not been removed. They occur with lesions of the nervous system at different anatomical levels from peripheral nerves to the cortex, and a study of the conditions in

which they arise illuminates the mechanisms underlying phantom limbs as a whole. The apparent rarity of cases is due to one important fact amongst others, namely, that the phantoms may exactly coincide with the real limbs. Even then they can be often separated and so revealed by active or passive displacement of the actual limbs from their resting position. On the other hand, under certain circumstances the dissociation of a real from a phantom limb may be invariable, so that the patient seems continuously to have a duplicated limb.

(*a*) *Peripheral nerves.*—As the result of lesions of peripheral nerves phantoms are comparatively rare and are confined to representation of those parts in which sensibility is abolished. Thus, in regard to individual nerves, they are confined to complete lesions of the ulnar nerve above the origin of the dorsal cutaneous branch, of the sciatic and, perhaps, of the trigeminal. In the case of the ulnar, a phantom, when present, is confined to the little finger in which alone, apart from the ulnar border of the hand, there may be complete sensory loss. When there is a phantom, abnormal sensations are referred to it, and since the phantom and the real fingers are usually in alignment, diagnosis of the severity of the lesion may then be difficult.

How the little finger, when deprived of sensibility, may be shed is well illustrated in the following case described by Head. A man who had received gross injuries to his elbow, including destruction of the ulnar nerve, later had to have his upper limb amputated. In his phantom hand, his thumb and fingers were fully represented except his little finger, which was completely absent.

The dysæsthesiæ and pain referred to the trigeminal area, which sometimes form distressing sequelæ of otherwise successful destruction of the gasserian ganglion with alcohol, may be possibly looked upon as a phantom half-face associated with complete sensory loss.

(*b*) *Plexus.*—I do not know of any case of phantom limb following a lesion of the lumbosacral plexus, but have examples after extensive damage of the brachial plexus. A typical case associated with pain in the hand and forearm may be quoted. It is that of a boy of 18, unusually intelligent and emotionally well balanced, who as the result of extensive traumatic damage to his right brachial plexus was completely paralysed in his upper limb, except for adduction at the shoulder, with loss of all forms of sensibility at and below the elbow-joint. The area of cutaneous sensory loss also extended along the outer aspect of the upper arm.

Thus, from the elbow downwards there was no evidence of either

motor or sensory function, but nevertheless he was aware of his fingers, hand, wrist and forearm, and he declared that he could move them to some extent. He suffered from paroxysmal pains which shot up his limb to his shoulder from his hand and wrist, in which they were most severe. This patient, in fact, had painful phantom fingers, hand and forearm, the component parts of which were exactly in alignment with those of his real limb.

Following exploration of the brachial plexus six weeks after the injury, the painful phantom persisted, but became separated from the actual limb, which he now knew to be there only by looking at it and touching it with his good hand or feeling it against his body. The phantom occupied a position in front of his chest, and failed to follow movement of the real limb when it was passively displaced at the shoulder-joint. The posture of the fingers and thumb of the phantom remained an exact reproduction of that of the actual hand.

A year after the original injury, the limb was amputated near the shoulder without influence on the phantom except that with progressive diminution of the pain it gradually approached the position in which the elbow would have been, and with return of postural sensibility in the shoulder-joint it moved with the stump. This shortening would temporarily increase during a bout of severe pain, an exception to the general rule. He noticed, however, that if he concentrated intently on some problem, the phantom would recede from the stump.

This case shows all the characteristics of a painful phantom which at first was exactly incorporated in the real forearm and hand. The effect of further interference with the plexus was to detach completely the phantom *along with the pain referred to it* from the paralysed and insensitive forearm and hand which still remained. The subsequent amputation did not result in much further change. But with recovery of postural sensibility at the shoulder-joint, the phantom again became attached to what remained of the arm.

(*c*) *Spinal roots.*—In some cases of complete loss of postural sensibility in all joints of a limb, as a result of severe lesions of posterior roots, a phantom limb is described. Thus, a patient with advanced tabes when asked to get up, said that he would try to do so if he could find his real legs. When at rest with his legs stretched out in bed he appeared to have one pair of legs—his real ones, but as soon as he moved a lower limb he became aware of a phantom leg.

The same experience was described by a patient with hypertrophic polyneuritis in whom there was complete absence of postural sensibility

in his lower limbs but incomplete paralysis and cutaneous sensory loss. In this case there was probably also damage to the posterior columns from compression by the thickened posterior spinal roots.

(*d*) *Complete division of the spinal cord.*—Phantom lower limbs following spinal transection were recorded during the last Great War [12]. For a few weeks after his injury the patient feels as if the part of his body, including the lower limbs, below the level corresponding to the lesion, is gone. With the return of reflex activity, however, he usually says that he is once more aware of his lower extremities and trunk. These phantoms commonly consist of thighs and feet, the legs between the knees and ankles being absent, but occasionally the calves are also represented. For the first few months at least, the most common posture is one of flexion, which may be extreme, so that the phantom lower limbs seem to be completely folded up with the knees against the abdomen and the calves resting on the backs of his thighs. In other cases, the legs appear to be tucked under him with the heels in contact with the buttocks, as if squatting, or one limb may be crossed over the other. Many variations of these postures are described, but the lower limbs are invariably more or less bent, although the actual limbs are lying extended on the bed. As reflex activity increases and spasticity of the flexor muscles develops, the real limbs also come to adopt a somewhat flexed position at rest and are subject to flexor spasms, which may be violent. But the phantom limbs never "jump," nor can their position be influenced by voluntary effort, although it often varies to some extent from day to day for no apparent reason. Variation in the clarity of the phantom limbs is, however, influenced by the patients' general health. For example, during a febrile attack the phantoms may become faint, and after the fever has passed become clear again. In addition, dissociation of the illusionary and the actual limbs may in time disappear.

A variety of aches and discomforts may be referred to the phantom limbs and are apt to be more intense with low than with high spinal lesions and, rarely, pain may be described. Thus, a patient with a complete lesion at the level of the ninth thoracic segment said "I feel as if someone were pulling at my legs trying to get them straight. The more they pull the tighter the strings become and soon I have to cry out with pain." He also complained of a girdle sensation round his buttocks and upper parts of his thighs and testicles which at times was severe enough to be painful. But discomfort rather than pain is more

common, and the most frequent complaint is one of tiredness and tightness of the phantom limbs.

(*e*) *Posterior columns.*—That abolition of all spinal function below the level of a transverse lesion is unnecessary for the appearance of phantom limbs is demonstrated by cases in which the sensory loss is more or less confined to postural and allied forms of sensation. The brunt of the lesion has then fallen on the posterior columns. An excellent example of a phantom lower limb under such circumstances is the following case. A man aged 57 had a severe attack of herpes zoster with the rash in the distribution of the fourth thoracic dermatome on the left side. Myelitis developed at the corresponding spinal level and a few segments above it, giving rise to weakness without spasticity of his lower limbs, left more than right, and of his trunk below the middle of his chest, along with the appropriate reflex changes and hesitancy of micturition. The sensory alterations were interesting. There was cutaneous tenderness on the inner aspect of both upper arms and on his chest in front and behind below the second ribs in a band about 2 in. broad. Below this level, light touches and pin-pricks were everywhere appreciated and correctly localized, but on the left side, and in particular in the lower limb, there was over-reaction to both forms of stimulation, especially to pin-prick. On the right side, sensibility to prick was diminished below the knee. Recognition of posture and passive movement was, however, completely abolished in his left lower limb, including the hip-joint, but was only impaired in the right lower limb. Vibratory sense also was absent on the left side below the rash and diminished on the right.

From the time of onset of his spinal symptoms, he was conscious of a phantom left lower limb. Both real lower limbs at rest were habitually extended and were not disturbed by involuntary movements. The phantom extremity, however, always appeared to be bent in a natural position with the knee raised and the sole of the foot resting on the bed. When he was propped up, the phantom foot seemed to go through the mattress and to protrude under the bed, the flexed position of the phantom limb remaining unaltered. He had no pain or discomfort in it, and he was unable to move it, but it seemed to him to be very real. No other change in posture was observed by the patient until, with recovery of postural sensibility, the phantom gradually became less flexed and finally merged into his real limb. This case clearly demonstrates the direct connection between separation of the phantom from the real limb as well as immobility of it, and postural sensory loss.

Similar examples of phantom upper or lower limbs are sometimes described by patients with disseminated sclerosis when the posterior columns are so severely affected as to give rise to complete postural sensory loss at all joints. Reduction of the other forms of sensation appears to be unimportant.

(*f*) *Subcortical cerebral lesions.*—I do not know of any example of phantom limb associated with a lesion of the brain-stem. The following, however, is a brief account of an excellent case of Van Bogaert's [17] of a phantom upper limb following an infarct which involved the optic thalamus and internal capsule as confirmed by autopsy. A man aged 60, with cardiovascular and renal disease, had a stroke which resulted in right hemiplegia with complete paralysis of the upper limb, but with involuntary athetoid movements, gross postural sensory loss, severe pain and over-reaction to cold. If he was wakened in the night by a spasm of pain, he often felt as if his right arm were being twisted and crushed under his head and would cry out to a neighbouring patient or the nurse to pull it down. He was able, to some extent, to correct this erroneous impression if he touched, with his left hand, his paralysed arm which was lying stretched out beside him, and also, by looking at it, if the ward were illuminated.

A similar case had been previously described by Pineas [11] of right hemiplegia in which the patient imagined that her paralysed arm was lying across her chest when it was actually on the bed alongside her trunk. But, unlike Van Bogaert's patient, the illusion was not dispelled by touching the paralysed limb with her sound hand. One difficulty, however, in the evaluation of evidence in cerebral cases of this kind is in regard to the associated mental state, which in arteriosclerotic subjects is often impaired.

(*g*) *Sensory focal epilepsy.*—To embark upon a description of the various disorders of the recognition of body shape from disturbance of function of the parietal sensory cortex would out-limit the scope of this lecture; for, in the main, they deal with the effects of disintegration at higher levels. I shall, therefore, confine myself to those which bear upon our immediate problem except to mention the pertinent syndrome described by Babinski [1], who named it "anosognosia," in which the patient, hemiplegic from a cerebral lesion, usually right-sided, is unaware of his paralysed limbs. The interpretation of this phenomenon has been much discussed, but need not now detain us. Let me at once give the main features in three cases of sensory epileptiform attacks, which illuminate the questions we are considering.

A phantom hand or foot along with loss of postural sensibility in the real limb may be an associated transient manifestation of a sensory Jacksonian epileptic attack. Thus a woman with cortical atrophy of the right parietal lobe, as found at operation, was subject to sensory fits which always commenced in her left foot. During and, for a short time, after each attack she felt as if she had two sets of toes on the left side, the phantom toes being curled down towards the sole of her foot. For so long only as the phantom persisted, there was complete loss of postural sensibility in her toes. In a second case, in which the nature of the parietal cortical lesion was unproven, the patient in, and following, each Jacksonian attack experienced sensations as if her right forearm had disappeared and her hand were attached to her elbow. This illusion, and the loss of postural sense which was confined to the fingers, wrist and elbow, were again transient. It calls to mind the shortening of a painless phantom limb after amputation, which although usually gradual, is sometimes immediate, so that the patient from the first feels as if his phantom hand or foot is attached to the end of his stump. In such cases postural sensibility is preserved in the stump whatever its length.

That a phantom representative of a *complete* limb may be a manifestation of a brief cortical, cerebral seizure is shown by the following case. A retired policeman, aged 63, had for years been troubled with attacks of a phantom left upper limb. Each attack was sudden in onset, lasting only a few seconds, and in it he had no knowledge, except from vision, of his real limb, but was aware of a phantom arm which was in some other position, usually to the right. If he happened to be holding something in his left hand, just before the attack, he would drop it, so that postural sensibility may have been temporarily lost. He was an arteriosclerotic with hypertension, but although he was generally weak and had bilateral extensor plantar responses, there was no local paralysis of his left upper limb. His memory was good when I first saw him and he appeared to be mentally normal. Later, however, when in the London Hospital, he was for a time difficult and suspicious.

The attacks in this case were again undoubtedly due to a local functional disturbance of the sensory cerebral cortex and are of especial interest in that they combined loss of recognition from somatic impressions of the affected limb with a phantom representation which was abnormally projected. It is in fact an example of local anosognosia with a phantom. Head and Holmes' [5] case already quoted of disappearance of a post-amputation phantom foot following a lesion of the opposite parietal cortex

is complementary, and it is regrettable that full details of it are not available.

The delimitation of the lesion within the sensory cortex in these cases is unknown.

## An Interpretation of these Phenomena.

(1) *Sensory functions which subserve recognition of body shape.*— Before embarking upon an analysis and explanation of the phantoms just described it would be helpful briefly to consider, from the physiological approach, the means by which the shape of the body is recognized. This can be considered from a sensory or motor point of view, but only in an artificial way, for it is essentially a combined sensorimotor function. As with all neural activity, its aim is skilful, purposive action. It begins in infancy and continues, almost unremittingly in the waking state throughout life, as a natural urge, under conscious guidance, to perfect the use of the body and its limbs for instinctive needs as well as occupational and recreational pursuits. In other words, expert use of the body is the result of trained effort.

At the same time, all individuals do not start at birth with equal endowment. Some have greater energy and desire to strive than others; potentiality and realization of need for useful adaptation are inconstant and the physical mechanisms, through which these mental functions operate, are also variable. This variation in the inborn perfection of the functional machine is commonly recognized and is expressed by such terms as having a good eye for games, which means perfect co-ordination of the body as a whole in action, under the stimulus of vision in relation to external objects. But the same smooth and well-ordered use of the body can be achieved by the congenitally blind, although the externally projected guidance is then through touch, hearing and smell. A common endowment in such gifted persons is natural strong right- or left-handedness. Those individuals who are neither right- nor left-handed, and, still more, those left-handers who, through the misguided efforts of their parents and teachers, have been unable to resist the compulsion to become right-handed, never become expert at games or other complex activities, whatever their mental equipment may be. They are handicapped and remain throughout life more or less clumsy not only with their limbs but often in articulation, for many of them are stammerers. They stumble with their tongues as well as with their limbs because the background of their actions is not automatic and so sufficiently "thoughtless." Their development has been ataxic because it has involved indecision from the absence of clear leadership from one or other hand.

Now competency in action means competency in the recognition of shape and postural change of the body. Its component parts are not uniform in function, for the limbs, especially the upper and in particular the leading limb, are essentially organs of movement, whilst the trunk more often behaves posturally and its reactions are secondary to those of the limbs and head. That is to say, the trunk is less used voluntarily than are the head and limbs, especially the upper. The arms are, in fact, pre-eminently organs for movement and exploration, the fingers being tentacles on jointed stems. The lower limbs are also more movable organs functionally than is the trunk, but are less continuously the centre of conscious direction than are the upper limbs, especially the leading one. Further, no part of the body in action is ever entirely independent of the whole. Independence is relative, especially in regard to the limbs. Each upper limb has more functional individuality than a lower limb, and still more a finger than a toe. But every movement, however local and fine, is accompanied by adjustments of a neighbouring segment and, in more vigorous actions, of the whole body.

The sensory functions concerned in the control of movements and postural reactions are those which underlie the recognition of body shape. They are forms of sensation which are projected, namely, postural sensibility, tactile localization and vision. The sensations of touch, pain and thermal change are the media with which localization works, touch being more constantly in use than the others. Of these three forms of projectional sensation, vision alone is unessential for postural recognition. This view has been opposed by Van Bogaert [17], Lhermitte [6] and Schilder [15], who maintain that the visual image of the body should be given first place, and that it plays an important part in the persistence of amputated limbs as phantoms. Many arguments can be used against them, but the most convincing of all is that provided by the expert use of the body and perfect postural recognition of the congenitally blind.

Where vision is decisively brought into play is in relating the body to external objects. It is the chief sensory function by which projection is carried a step further, from acquaintance with the surface and posture of the body to knowledge of the environment.

According to Head and Holmes [5], the standard by which changes in the outline of the body and postural variations are measured and are what they termed "schemas" or "plastic models." They are constructed throughout the life of the individual from two main sources: (1) Sensory impulses derived from receptors chiefly in joints, muscles and tendons, and (2) from sensory impressions from the skin and subcutaneous tissues

which subserve recognition of the spot stimulated. The first is concerned with postural sensibility and the second with localization and shape. Each fresh set of impulses originating from these sources are, through a process of integration, brought into relationship with physiological schemas already constructed. "By means of perpetual alterations in position we are always building up a new model of ourselves which constantly changes. Every new posture or movement is recorded on this plastic schema and the activity of the cortex brings every fresh group of sensations evoked by altered posture into relation with it. Immediate postural recognition follows as soon as the relation is complete" [5].

In the same way, outline schemas, or plastic models of body shape, are constructed through the process of localization of the innumerable tactile, painful and thermal impressions which arise from stimulation of the surface of the body.

The recognition of body shape and changing posture is thus achieved through the activity of physiological mechanisms by means of which three main "plastic models" are constructed—namely, surface, postural and visual. Normally, they act together and in complete co-operation, but their separate reality is laid bare by the dissociations which follow injury to their underlying sensory mechanisms. Of the three, the visual image is not essential for postural and surface recognition and so for the development of the continually changing model of the body, although it is normally employed as an important adjuvant. Its prime function is to bring the body into relation with its environment.

(2) *The pathological physiology of phantom limbs.*—Why should the postural and surface models remain in many respects unchanged when a limb is amputated? And why should a limb or other part of the body which is simply deprived of certain forms of sensation, whether from peripheral or central lesions, appear to be reduplicated? Why pain in a phantom part? What is the meaning of voluntary and associated movement of the phantom? What explanation can be given for variation in position of the phantom, for its separation from the real limb under certain circumstances and for reduction or abolition of movement of it? Why, in certain cases, should a phantom part persist indefinitely and in others never develop? These are some of the questions we must attempt to answer.

Let us first consider the case of the phantom limb following amputation. Stimulation by the processes of healing of the proximal ends of the divided nerves evokes sensations which are projected and interpreted as if the limb were still present. As has been said, they are never quite

normal. These paræsthesiæ, through simultaneous excitation of the schema underlying tactile localization and shape are projected and animate the surface or outline model of the absent part. Similarly, irritation of fibres concerned with postural sensibility give rise to impulses which help to keep alive the postural model, so that the phantom is correctly placed and moves with the stump. These sensations, in the absence of pain, are, however, weak, so that, as a rule, only the peripheral segments, the hand or foot, which are most richly endowed with sensory end-organs and fibres, are represented in the phantom. Retention of the phantom is in part due to the abnormal qualities of the tactile and other sensations, in spite of their relative weakness and the antagonistic evidence from visual and other senses. During the stabilizing process of healing of the divided nerves, sensory impulses diminish, and sensations become correspondingly fainter, with the dual result that the phantom is increasingly less obvious in outline and projection of it is defective. In consequence, it gradually approaches the stump, into which it finally disappears and fades away. A new shape of the body is now accepted. In other words, there is no longer a conflict in evidence from the patient's senses. If, however, the phantom is painful, which is usually the result of grossly abnormal conditions in the stump, the phantom may persist indefinitely and retain its original position. Further, the hand and fingers are not only much more obtrusive and clearly defined, but more of the amputated part is represented by it. Voluntary movement is restricted or impossible because of aggravation of pain. When, however, pain in the phantom is successfully abolished by lateral chordotomy or early removal of abnormalities in the stump, the phantom may behave as if it had been painless from the first. So long as it persists, the patient may say that he can move the fingers more or less freely, and in time it, as a rule, becomes dim, retreats to the stump, and finally vanishes. Thus, the main effects of pain on the phantom are to limit voluntary movement of it. But it does not separate the phantom from the stump.

Dissociation of the phantom and stump in movement, irrespective of whether the phantom is painless or painful, can be achieved only by the complete abolition of postural sensibility in all joints of the stump, as by posterior root section. The position of the phantom may or may not be altered by the operation, but it will be uninfluenced by either active or passive displacement of the stump. Another important effect of postural loss in the stump is great limitation or even absence of voluntary movement of the phantom fingers, whilst the clarity of the phantom outline may be unimpaired. Should postural sense at the shoulder-joint be only

temporarily lost, as from disturbance but not destruction of the brachial plexus, with its return the phantom regains its previous connection with the stump so far as movement is concerned.

From these findings certain conclusions can be drawn: (1) The parts which are usually represented in a painless, post-amputation phantom are peripheral—hand, foot, nipple, glans, tip of nose. They are the parts which anatomically are most heavily endowed with sensory end-organs and, physiologically, are most represented in the model of body shape. (2) Pain in the phantom is due to excessive stimulation of the nerves in the stump and results in extended representation of the absent limb in the phantom and limits voluntary movement of the phantom only because voluntary effort aggravates the pain. When pain is abolished, voluntary movements are then as possible as if the phantom had from the beginning been painless. (3) The prevailing posture of the phantom is that of the part at the time of amputation. It is as if the postural model had become frozen when normal stimulation ceased. (4) Voluntary movement of painless phantom fingers or toes is usually possible and often considerable. One of the main factors upon which it appears to depend is preservation of postural sensibility in the stump. (5) So long as sensation in the stump is intact, the phantom remains an integral, if modified, part of the models of body shape and position. Abolition of postural sensibility results in a dissociation, in that whilst the surface or shape model of the phantom remains unaffected (whether painless or painful), the postural model of the phantom is in certain respects destroyed. For the state of affairs now is as follows. Because of the persistence of the paræsthesiæ and of their projection, from preservation of the sensory functions underlying localization and shape, the outline model of the phantom part remains. In consequence, it is still placed in relation to the body, and when the whole body is moved the phantom moves with it. But it no longer is connected with the limb which it represents, for its position is now unaltered by movement of the stump. It is as if it were suspended in mid-air, in part a foreign body which cannot be got rid of because of preservation of its connection with the general surface-shape model. Further, another important change has occurred in that the phantom can no longer be moved voluntarily, or at least voluntary movement is much diminished and, in addition, it ceases to be linked with the corresponding limb on the opposite side in associated postural reactions.

These observations lead to the conclusion that the postural model of a limb is largely *a postural model of movement*, the model of static posture being linked also with the surface model.

Let us now consider how these conclusions apply to phantoms associated with intact limbs following peripheral or central lesions of the nervous system. In cases of extensive and severe damage to the brachial plexus, so long as postural sense in the shoulder remains, the phantom and the real limb tend to be in alignment and movement at the shoulder-joint displaces both together as one. In other words, the surface and postural modes are superimposed and correspond to the intact limb. At the same time the phantom fingers and hand can be separated from the real parts, for the time being, by voluntary effort. Disappearance of the postural movement model, however, follows loss of postural sensibility in the shoulder, the phantom becomes separated from the real limb and its position, which is now largely stationary, is uninfluenced by active or passive movements of the stump. Further, when the phantom has been painful the pain is still referred to it in its new position and not to the intact limb, so that pain is linked with the surface-shape model and is uninfluenced by loss of the postural-movement model.

The same dissociation of the components of the phantom limb schemas is found when there is complete postural sensory loss in the limb resulting from lesions of the posterior roots or columns. In such cases, other forms of sensation may be largely retained. As with peripheral lesions, the phantom limb remains attached to the body as a parasite because of the surface model, but it no longer possesses independent movement, and in this sense has ceased to behave like a jointed limb. It has lost its connection with the postural-movement model. The same detachment or dissociation applies to phantom limbs from total transverse spinal lesions and to the phantom arm which is occasionally associated with hemiplegia of cerebral origin, if loss of postural sense is severe. It is true that, in these central cases, the patient may be aware of some involuntary change in position of his phantom, but he has no power to bring it about himself. Further, as in spinal cases, the phantom appears to be separated from the limb which it falsely represents. A similar dissociation of limb models from loss of postural sensibility, but as a transient phenomenon, may form part of a Jacksonian sensory epileptiform attack, as is shown by the cases described earlier in this lecture.

On the other hand, destruction of the cortical sensory receptive mechanism in the parietal lobe, which is concerned with the development of postural and surface models and with the recognition of change, causes immediate abolition of the phantom limb.

I have frequently referred to voluntary and associated movements of phantom limbs and must now attempt to explain this interesting illusion.

There is little difficulty with associated movements, for they are really postural reactions determined by powerful voluntary movement of the opposite intact upper or lower limb as the case may be [13]. They represent reflex pattern adjustments for postural purposes which are largely independent of sensation and voluntary movement. The participation of the phantom hand in such reactions and, in particular, the evocation of them in the opposite intact limb by voluntary attempts to move the phantom, demonstrates the intimate linkage of the postural model of a limb with that of its fellow, and indeed of the body as a whole. An attempt to explain the illusion of direct voluntary movement of the phantom hand is fraught with greater difficulties, for the physiological processes which underlie the initiation of willed movements are imperfectly understood. It has been claimed that the illusion of movement of the hand is excited by activation of the muscles of the stump, but this argument breaks down in the case of short stumps. We have seen, however, that preservation of postural sensibility in the joints of the remaining portion of the limb has an important bearing on the problem. Abolition of postural sensibility by destroying the postural-movement model of the limb prevents the illusion of direct voluntary movement of the phantom. All that can be said for the present is that movement is initiated from the cortex, but for it to be possible the postural-movement model of the limb must be intact, which necessitates preservation of postural sensibility in the stump.

Lastly, it is necessary to consider why phantoms are inconstant not only in their appearance but also in their disappearance. The answer lies in the interaction of two processes at once complementary and antagonistic, namely, peripheral stimulation and central inhibition. No one can deny the importance of excitation of sensory fibres of the divided nerves in the cases of painless phantoms of normal duration and of painful phantoms. Does, however, peripheral stimulation still continue in the case of persistent, painless phantoms when the stump has healed and no tender abnormalities are present; and still more, does it play a part in phantom limbs following central lesions? I maintain that it must, if for this reason alone: that the sensations referred to the phantom are always more or less abnormal. The problem is essentially the same as that of pain of central origin, which has been discussed elsewhere [14]. We are ignorant of the means by which such stimulation can occur, but local chemical and circulatory changes acting upon peripheral or central sensory fibres in a pathological condition of excitability are not inconceivable.

But, as in all neural activities, excitation cannot operate apart from its

twin process, inhibition. Psycho-physiological inhibition must, quite apart from the degree and duration of peripheral stimulation, influence the development and persistence of phantom parts. Its strength, however, is again variable and bears direct relationship to the adaptive capacities of the individual. When it is strong and peripheral stimulation is slight, whatever level the lesion may be, either no phantom develops or it is transient. Under this category are to be grouped "anosognosia" of Babinski and cases of amputation without or with a temporary phantom, as well as the rare examples of tender stumps without phantoms. Massive painful stimulation from abnormalities in the stump is always able to overcome central inhibition and the phantom remains for so long as pain is referred to it. When psycho-physiological inhibition, and so adaptability, is subnormal, even a painless phantom part may last indefinitely; and, on the other hand, inhibition may be temporarily reduced by ill-health or emotional disturbance with resultant reappearance of a phantom that had gone. Thus, the two forces excitation and inhibition are in continual interaction in the attempt to establish a new body shape.

In conclusion, contemplation of the story of the phantom limb forces the realization of the striking contrast presented by the difficulty which the individual has in accepting, physiologically as well as psychologically, his shortened limb and his possession of a strong natural aptitude to elongate his limbs at will in the use of tools. When, for example, he is (after amputation) wearing his artificial limb he feels with its foot and can distinguish between irregularities on the ground. There is no essential difference between this achievement and that of the surgeon who projects his fingers to the end of his probe, or that of the edentulous who incorporates his artificial teeth. In the same way, the trained motorist identifies his car with himself, the airman with his machine and the angler with his fly at the end of a long line. Samuel Butler [2], who developed this idea, called tools detachable limbs and maintained that the capacity to employ them in this way is one of the main characteristics which distinguishes man from lower animals. The competency of this aptitude for projection varies in different individuals for reasons which are inborn as well as of training. But, strong as it is, it is at once inhibited if discomfort or pain is evoked by contact of the tool with the body. The dental plate or the artificial limb is immediately rejected as a foreign body if there is an ulcer on the gum or a tender neuroma at the end of the stump. It is another illustration of the dominance of pain over adaptive functions.

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# CEREBRAL ELECTRICAL CHANGES IN EXPERIMENTAL CONCUSSION.

BY DENIS WILLIAMS AND D. DENNY-BROWN

*(From the University Laboratory of Physiology, Oxford.)*

THE progressive changes in cerebral electrical activity resulting from general and local cerebral injury in man have recently been described (Williams, 1941). Repeated electro-encephalographic records were made through the intact skull in patients with injuries of varying severity, the shortest interval after injury being fifty minutes. Abnormally slow waves of electrical potential change were observed, the voltage, period and persistence of which were directly related to the severity of the injury and inversely proportional to the degree of recovery. Denny-Brown and Russell (1941) studied changes in reflex activity and in the vascular and cardio-respiratory mechanisms after producing concussion in animals under standard conditions. They found that the phenomenon of concussion was associated with complete abolition of reflex activity and loss of tone in all muscles. Inspiratory spasm, bradycardia and a rise in blood-pressure also occurred when concussion was produced, but these changes appeared to be epiphenomena caused by interference with medullary nuclei, but not causally related to the cessation of spontaneous and reflex movement or to the loss of consciousness characteristic of concussion. As a result of their observations, Denny-Brown and Russell consider that concussion is a state of direct traumatic central inactivity or paralysis. Although it was possible to make accurate observations on the functional activity of brain-stem structures, no similar observations could be made upon cerebral function. We have therefore proceeded to relate the immediate effects of experimental concussion upon these lower reflex functions and upon the electrical activity of the cerebral hemispheres. It was hoped that in this way further information upon the mechanisms underlying traumatic loss of consciousness might be gained and the observations made upon animals and man might be co-ordinated. It is the purpose of this paper to describe the method used and the results obtained.

## METHOD.

Cats were lightly anæsthetized by intraperitoneal veterinary nembutal (Abbott). In two instances light ether was also used during the operative procedure, but recording was not commenced until an hour had elapsed after the ether administration. In all cases the basal anæsthesia was sufficiently light to allow preservation of the corneal, pinna and head-shaking reflexes, and occasionally spontaneous licking and swallowing movements were also present.

The cranium was exposed through a longitudinal incision, the temporal muscles were reflected, and either one or two 2 mm. burr holes were made through each parietal bone from 10 to 15 mm. from the mid-line. Care was taken to preserve the dura and to prevent any intracranial damage. The burr holes were then threaded and a brass screw electrode 3 mm. long was screwed into the hole until the smooth rounded lower end was flush with the inner surface of the calvarium, thus re-establishing the continuity of the cranium. As the electrode was firmly fixed into the bone without any deformity to the cranium, local contusion of the cerebrum at the site of the electrode was obviated, and the contact between electrode and cranium was so intimate that in no instance was it disturbed by the concussive force used. The electrodes were then securely attached to fine shellac-coated copper wire which allowed full movement of the head. Indifferent electrodes consisted of a stainless steel needle in the base of each pinna. Respiration and heart rate were recorded from two similar electrodes in the thoracic and abdominal walls.

The cat was supported in a small fabric hammock suspended by six cords inside an earthed screen cage. The whole body and head rested in the quadripedal position, the limbs being passed through appropriate holes in the hammock. By this method artefacts caused by movement were minimized and the animal could be easily observed. The pairs of electrodes were connected to the inputs of a three-channel ink-writing electro-encephalograph, incorporating a push-pull circuit (Grass). The parietal and indifferent ear electrode of corresponding sides led to the grid and plate of two recording channels, and the chest and diaphragmatic electrodes to those of a third. Simultaneous records were made from both hemispheres under identical amplification and amplifier characteristics, time was marked in seconds, the recording speed being 1 in. per second. Calibration signals were recorded from 50 and 100 microvolts across the input electrodes at the end of each experiment (figs. 1-4).

At the beginning of the experiment the corneal and pinna reflexes were elicited and any spontaneous movements, especially of the tongue

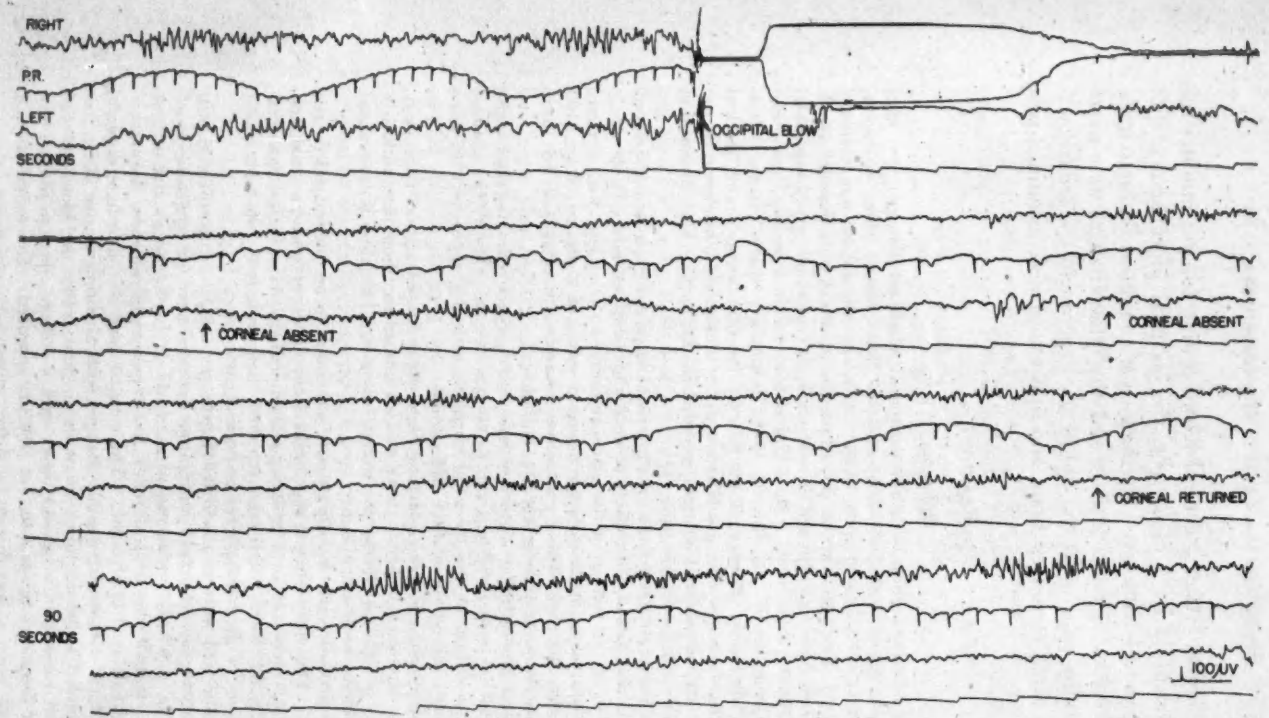


FIG. 1 (*Cat 1*).—*Generalized diminution in electrical activity with bradycardia, accompanying concussion.* In this and the following figures the records read from left to right and from above downwards. They are continuous unless a time interval is stated in the record. Electro-encephalograms are recorded on two writers and pulse-rate and respiration (P.R.) on the third. Time is in seconds in all cases. RIGHT represents an electro-encephalographic record obtained with an active electrode on the right parietal area and with an indifferent lead from the right ear. LEFT represents a record under similar circumstances from the left side. In this figure a hard occipital blow has caused an artefact in all leads which lasts for about ten seconds; this is followed by a diminution in activity from both hemispheres. This diminution is general and symmetrical and is accompanied by bradycardia and alteration in respiratory rhythm. The rhythmic outbursts of relatively fast electrical activity are due to the nembutal anæsthesia. About 140 seconds after the head injury the electrical activity has begun to return. At this time there is some cardiac irregularity although the rate is again increasing. The corneal reflex returned fifty seconds after the blow.

and tail, were noted. After a control record lasting for five minutes the animal was concussed by a blow upon the occipital protuberance with a light, long-handled hammer. The e.e.g. was continued without interruption and the state of reflex activity and any other changes were noted upon the recording paper. At the end of the experiment the animal was killed and the skull, meninges, brain and upper cord were examined post mortem.

## Results.

### *Protocols.*

*Cat 1.*—After anæsthetization with intraperitoneal nembutal 0·02 grm. per kilo, the corneal and pinna reflexes were retained but there were no spontaneous movements, and for the purpose of the experiment the animal was relatively deeply anæsthetized. The experimental procedure was identical with that described. Records taken from both hemispheres showed changes typical of barbiturate basal anæsthesia (Derbyshire *et al.* 1936). An occipital blow resulted in the phenomena of concussion (Denny-Brown and Russell, 1941), inspiratory spasm lasting fifteen seconds, general flaccidity, absence of all reflexes for fifty seconds and marked bradycardia. These changes were associated with an immediate diminution in the voltage of the electrical potential changes recorded in the e.e.g. This diminution affected all the frequencies observed (fig. 1).

*Blow 2*, given five minutes after the first, produced inspiratory spasm and delayed slight bradycardia and loss of the corneal reflex for fifty-two seconds. There was a further diminution in the voltage of the waves recorded on the e.e.g., with appearance of short runs of abnormally slow two-a-second waves of medium voltage (100 $\mu$v) 120 seconds after the blow. These gradually increased in number and size until the record was discontinued 15 minutes after the blow.

*Blow 3* produced similar bulbar signs and loss of the corneal reflex for sixty-eight seconds with a fall in pulse-rate from 200 to 60 per minute. There was again a diminution in the voltage of the electrical changes, with the appearance of runs of low voltage two-a-second waves after 200 seconds.

*Blow 4* was interesting. It was a glancing blow which caused a short inspiratory gasp without loss of corneal reflex. There was an immediate short diminution in electrical activity which only lasted for twenty seconds. The slow waves caused by the previous blows were still evident.

*At autopsy.*—Macroscopically the dura mater was found to be undamaged by the screw electrodes. The hemispheres and brain stem appeared quite normal and section showed no evidence of local bruising or hæmorrhage. Histological examination was made by Dr. Robb-Smith who reported that no lesions could be found within the substance of the hemispheres or brain stem.

*Cat 2.*—Anæsthetized with nembutal 0·019 grm. per kilo, the experimental procedure being unchanged. Corneal and pinna reflexes were present and spontaneous swallowing and licking movements occurred. A hard blow on the occiput caused flaccidity, loss of the corneal reflex for twenty-eight seconds, inspiratory spasm and a slight delayed bradycardia. The e.e.g. showed an immediate diminution in voltage of the electrical potential changes with abolition of the faster frequencies. The record remained much more stable than before the blow and 120 seconds afterwards runs of two-a-second waves with a voltage equal to that of the resting dominant frequency of six-a-second (150 $\mu$v) appeared. The record showed no further change when it was discontinued five minutes later.

*Blow 2.*—A very hard occipital blow resulted in inspiratory spasm, loss of all reflexes and muscular flaccidity followed by brief quadripedal running movements and then cessation of all activity. Immediately after the record there was a great reduction in the voltage of all frequencies in the e.e.g. which rapidly progressed until the record became quite flat. The heart beat became irregular, very slow and finally stopped.

*Autopsy* showed an annular fracture around the foramen magnum with forward displacement of the skull. There was contusion of the medulla at the site of fracture with extensive subarachnoid bleeding. The cerebral hemispheres were normal. Histological examination (Dr. Robb-Smith) showed that the cerebral hemispheres were apparently normal. There was a large laceration of the anterior portion of the medulla, with intramedullary hæmorrhage.

*Cat 3* was anæsthetized with intraperitoneal nembutal 0·016 grm. per kilo, light ether being used during insertion of the screw electrodes. The experimental procedure has been described. The corneal and pinna reflexes were present and brisk, spontaneous licking and swallowing and lateral tail movements were present. There were also occasional spontaneous leg movements.

*Blow 1* was subconcussive. There was a slight change in respiratory rhythm but the pulse and reflex activity did not change. The electro-encephalogram showed no change (fig. 2).

*Blow 2*, an occipital blow, caused slight bradycardia and respiratory spasm lasting four seconds, the pinna reflex was lost for seven seconds, and there was a momentary loss of muscular tone. Spontaneous movements were less active after the blow. The e.e.g. showed an immediate and remarkable loss of all fast activity which persisted until 170 seconds afterwards, when short runs of two-a-second waves of 100 $\mu$v. appeared. These persisted and gradually became more numerous and larger in size (fig. 2).

*Blow 3*, ten minutes after the first, resulted in mild concussion with loss of the pinna reflex for twenty seconds, inspiratory spasm for five seconds and a very slow pulse after ten seconds. The electro-encephalographic record again became relatively flat, none of the faster components being seen. The response was not materially different from the record after the previous blow.

*Blow 4* resulted in very mild concussion. There was respiratory spasm for four seconds, with bradycardia after six seconds. Again the e.e.g. flattened out immediately with loss of all fast activity. The electrical changes remained in abeyance until 158 seconds after the blow, when short runs of low voltage (50-100 $\mu$v.) sine waves with a period of approximately two-a-second appeared.

*Blow 5* was harder than the previous one. There was a prolonged inspiratory spasm with bradycardia after four seconds. The pinna reflex was absent for four minutes. There was an immediate and profound diminution in the electrical activity recorded in the e.e.g. and fast activity was practically absent. Slow waves with a frequency of one to two a second appeared 160 seconds after the injury.

*At autopsy* no fracture was found in the calvarium and the dura mater was normal. There was a small area of thin basal subarachnoid hæmorrhage, and a minute epidural clot under each screw. Except for a small hyperæmic area at the right occipital pole, the cerebral hemispheres were macroscopically normal (fig. 5). Histological examination was undertaken by Dr. Robb-Smith. There was no evidence of any intracerebral abnormality.

*Cat 4* was anæsthetized with intraperitoneal nembutal, 0·016 grm. per kilo. The corneal and pinna reflexes were present, and spontaneous licking, tail movements and even occasional leg movements were present. An intratracheal cannula was inserted, reflex coughing being very active, but otherwise the experimental procedure was unchanged.

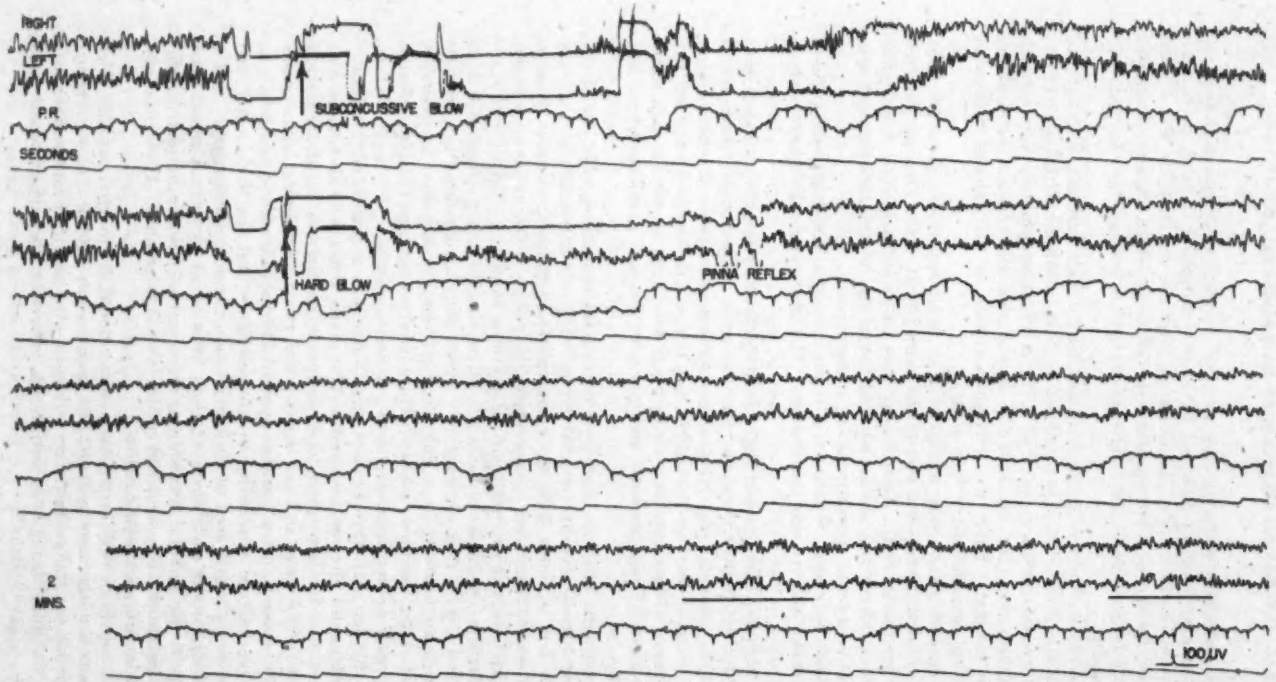


FIG. 2 (*Cat 3*).—*Difference between the effects of concussive and sub-concussive blows.* The conditions of recording are identical with fig. 1, except that pulse and respiration (P.R.) are recorded on the third channel instead of the second. The first sections of record show that a moderately hard occipital blow which caused slight change in respiration, reflex activity or pulse-rate, caused no alteration in electrical activity. The lower records show the result of a hard blow in identical circumstances. After a short artefact there is diminution in the electrical activity of both hemispheres, associated with bradycardia. The diminution in activity persists and in the fourth strip of record, 150 seconds after the blow, low voltage two-a-second waves begin to make their appearance. These are underlined. The pinna reflex returned seven seconds after the blow, concussion therefore being momentary.

*Blow 1* on the occiput stilled the spontaneous limb movements. The corneal reflex was absent for six seconds. The electrical activity seen in the e.e.g. subsided immediately, the fast frequencies being especially diminished, and slow waves (about two a second) appeared after 180 seconds.

*Blow 2*, twelve minutes after the first concussion, caused no change in respiratory or cardiac rate, nor was the corneal reflex lost. There was diminution in the electrical activity similar to that already described. Abnormally slow waves appeared after 170 seconds. These persisted but began to diminish in size and number twenty minutes after they had begun. A respiratory pump was then used at fifteen cycles per minute through the remainder of the experiment, in order to abolish any changes which might be caused by traumatic respiratory irregularity.

*Blow 3*, administrated thirty-five minutes after blow 2, caused concussion with delayed bradycardia and loss of the corneal reflex for nineteen seconds (fig. 3). The respiratory rhythm was maintained by the pump. Again there was immediate cessation of fast activity, with reduction in voltage of the dominant frequencies and the occurrence of abnormally slow waves 150 seconds after the blow. These slow waves increased in number and again persisted (fig. 3). The reduction in voltage of the normal frequencies lasted for 20 minutes.

A screw 3 mm. longer than those used previously was now screwed into the left parietal burr hole so as to impinge upon the dura mater and indent the cerebral tissue. A light blow was applied to this screw without any evidence of concussion. There was an immediate diminution in the number and size of the faster waves on that side. Abnormally slow waves appeared in a short run after twenty-four seconds. The record from the right hemisphere showed no change. A further heavy blow applied to the long screw on the left side caused a small circular depressed fracture around the screw and drove the screw a little distance into the cranial cavity. There was again no evidence of cerebral concussion. There was immediate diminution of the electrical activity seen in the e.e.g. recorded from this screw electrode, abnormally slow two-a-second waves appearing twenty-seven seconds after the blow. The electro-encephalographic record from the right hemisphere still remained normal. On both these occasions no change in reflex activity or cardiac rate could be seen.

*At autopsy* a very small bruise of cerebral cortex about 2 mm. in diameter was found under the site of the long electrode, but otherwise the hemispheres and brain stem were quite normal (fig. 5). Dr. Robb-Smith examined the brain histologically and could find no intracerebral damage.

*Cat 5* was anæsthetized with intraperitoneal nembutal 0·016 grm. per kilo. As the basal anæsthetic was light, ether was used during the operative procedure. The conditions were identical with those in the earlier experiments and artificial respiration was not used. On this occasion two screws 1 cm. apart were inserted into the left parietal bone, one which was 3 mm. long just filled the burr hole, while the other, 6 mm. long, penetrated about 3 mm. into the cranial cavity. A single 3 mm. screw was used on the right side.

*Blow 1* was on the long electrode and resulted immediately in four high voltage one-a-second sine waves being recorded from that electrode. The e.e.g. was otherwise unchanged. The contralateral record showed no change. It is possible that these large waves may have been due to an artefact, but no such change was seen in the other leads. Respiration and pulse were unchanged, and there was no evidence of concussion.

*Blow 2* was a very hard one applied to the same electrode. It caused a depressed fracture, the screw and bone fragments being partly driven into the cranial cavity. The pulse slowed temporarily but the corneal reflex was not lost. There was

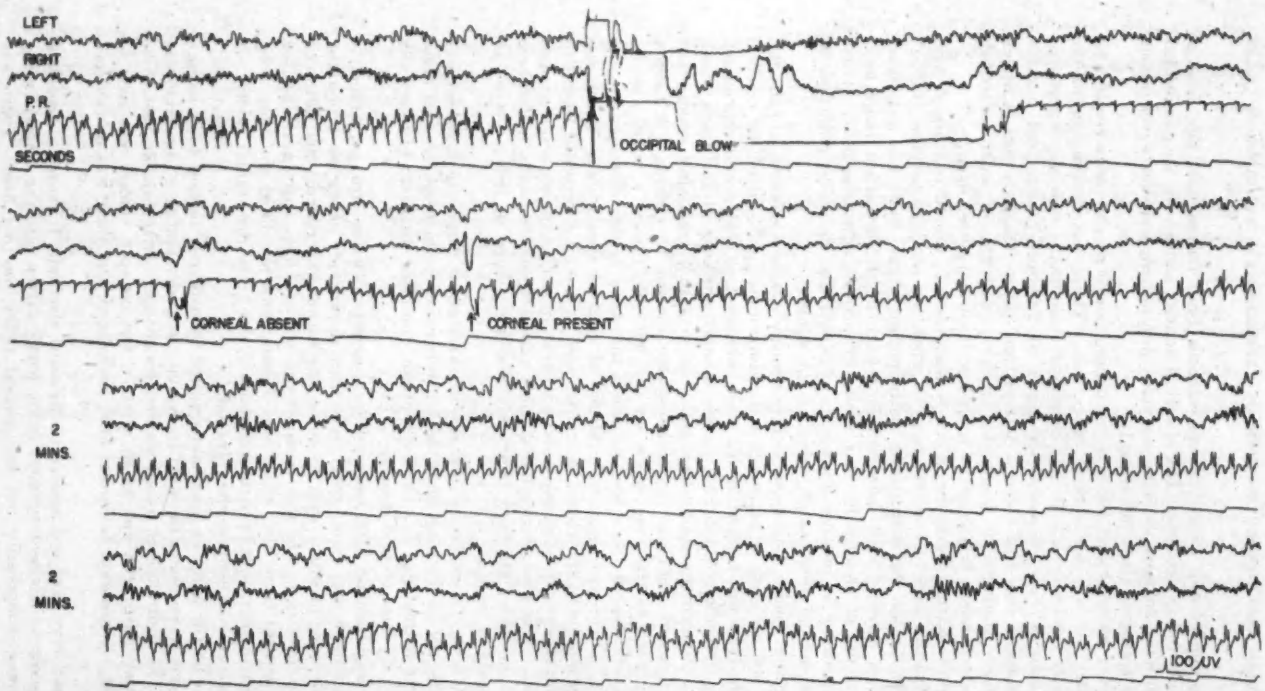


FIG. 3 (*Cat 4*).—*Diminution in electrical activity, and the appearance of slow activity occurring with concussion during artificial respiration.* Recording was identical with that in fig. 2, but the respiration was maintained artificially. After an occipital blow there was diminution in the electrical activity from both hemispheres. The corneal reflex was absent for twenty seconds and there was delayed bradycardia, followed two minutes later by an increase in pulse-rate. In the third record, two and a half minutes after the blow, rhythmical one-a-second waves make their appearance in both hemispheres, and two minutes later these waves have increased in number and voltage.

complete cessation of all electro-encephalographic activity under this electrode (fig. 4). The contralateral electrode and that on the same side 1 cm. away also gave normal records. The long screw was removed, as bleeding had been taking place from the fracture, but the dura was found to be dry and undamaged, so the electrode was re-inserted and once more no electrical activity could be recorded. Thirty-five minutes after the blow the electro-encephalogram recorded from the long screw electrode had returned to its former state (fig. 4).

*Blow 3.*—A 6 mm. electrode was then inserted into the anterior burr hole on that side, and it was struck lightly. There was now immediate and complete cessation of activity under that electrode, the contralateral hemisphere giving a normal e.e.g. Electrical activity began to return slowly and after fifty minutes the records from the left hemisphere showed a voltage change similar to that observed initially, but there was more slow activity and fewer fast frequencies.

*Blow 4.*—The left anterior electrode was tapped lightly an hour after the previous blow. No change was observed in the animal's condition or in the e.e.g. recorded from either hemisphere.

*Blow 5*, a harder blow, caused diminution in activity in the e.e.g. from that side only.

*Blow 6.*—A still harder blow twelve minutes afterwards caused the accompaniments of concussion already described. There was diminution in electrical activity from both hemispheres with the appearance of abnormally slow waves 205 seconds after the concussion. A stainless steel needle electrode inserted into the burr hole also showed the diminution in electrical activity, indicating that this was not due to any physical short circuiting effect of blood, or to a defect in the screw electrodes. After a further interval of about an hour the large screw was introduced into the right parietal bone and the whole procedure was repeated with similar results.

*At autopsy* a severe local contusion with destruction of cerebral cortex was found under the left posterior burr hole with a thin layer of subarachnoid bleeding. A minute petechial spot was seen under the corresponding point on the right side, where a small subdural hæmorrhage 1 mm. thick was found, and there was a very small contusion without hæmorrhage under the anterior electrode on the left side. The rest of the meninges, cerebrum and brain stem were normal (fig. 5). On section a fairly extensive hæmorrhage of traumatic type was present in the white matter of the left hemisphere. No hæmorrhages were present elsewhere and there were no fat emboli.

### Reduction in Fast Activity.

Reference to the protocols shows that in all instances where concussion resulted from a blow, there was an immediate reduction in the voltage of the cerebral electrical changes recorded in the e.e.g. This reduction was especially selective upon the faster frequencies, the fastest of which disappeared completely. A subconcussive blow, that is a blow which did not result in flaccidity and momentary loss of the corneal or pinna reflexes, was not accompanied by any such reduction in voltage, while severe blows resulting in prolonged loss of reflex activity caused almost complete disappearance of all frequencies of electrical potential change. All degrees of severity between these two extremes showed the same correlation (figs. 1 to 3).

The spindles of fast spikes caused by nembutal anæsthesia were

resistant to change, unless the animal was very lightly anæsthetized, but when ether was used for the surgical procedure, as in animals 3 and 5, and a lighter basal anæsthesia used, the concussive effects could be observed through the less marked anæsthetic effects. The reduction in fast frequencies was found to persist for more than twenty minutes after the blow (Cat 4) and it was observed to subside slowly.

### Slow Activity.

Abnormally slow waves, indistinguishable from those recorded in man (Williams, 1941 (*a*)) appeared at an inconstant interval after the injury. This interval ranged from 25 to 205 seconds. In all cases where concussion occurred, the time of onset was surprisingly constant. It was usually in the neighbourhood of three minutes (120-205 seconds). The presence of slow waves and their time of onset was less constant after local contusion. The frequency of the slow waves was approximately two a second, and they invariably appeared first in short runs of three or four with long intervals which became shorter as the voltage and number of the slow waves increased. These changes occurred in cats whose brains did not show any histological changes post mortem.

### Reflex Activity.

The corneal and pinna reflexes invariably recovered before any return in electrical activity was observed or before the abnormally slow waves appeared in the e.e.g. The discrepancy was often great, but animals which had shown spontaneous movements remained quiet during the period of reduced electrical activity and of slow wave formations.

### Visceral Effects.

It has already been stated that there was no constant relationship between the duration of inspiratory spasm, the degree of bradycardia, or its time of onset, and the amount of change seen in the e.e.g. The results in Cat 4 show that artificial maintenance of respiration at a constant level does not affect the decrease in electrical activity or the appearance of slow waves, so that alteration in the blood $O_2$ or $CO_2$ content is not responsible for the changes observed in the e.e.g. Figs. 2 and 3 show that the diminution in electrical activity occurs before the bradycardia, and abnormally slow waves may appear in the absence of any significant cardiac slowing.

### Focal Damage.

Percussion upon a screw impinging on the cortex caused changes in the e.e.g. recorded from that area immediately below the screw (Cats 4 and 5).

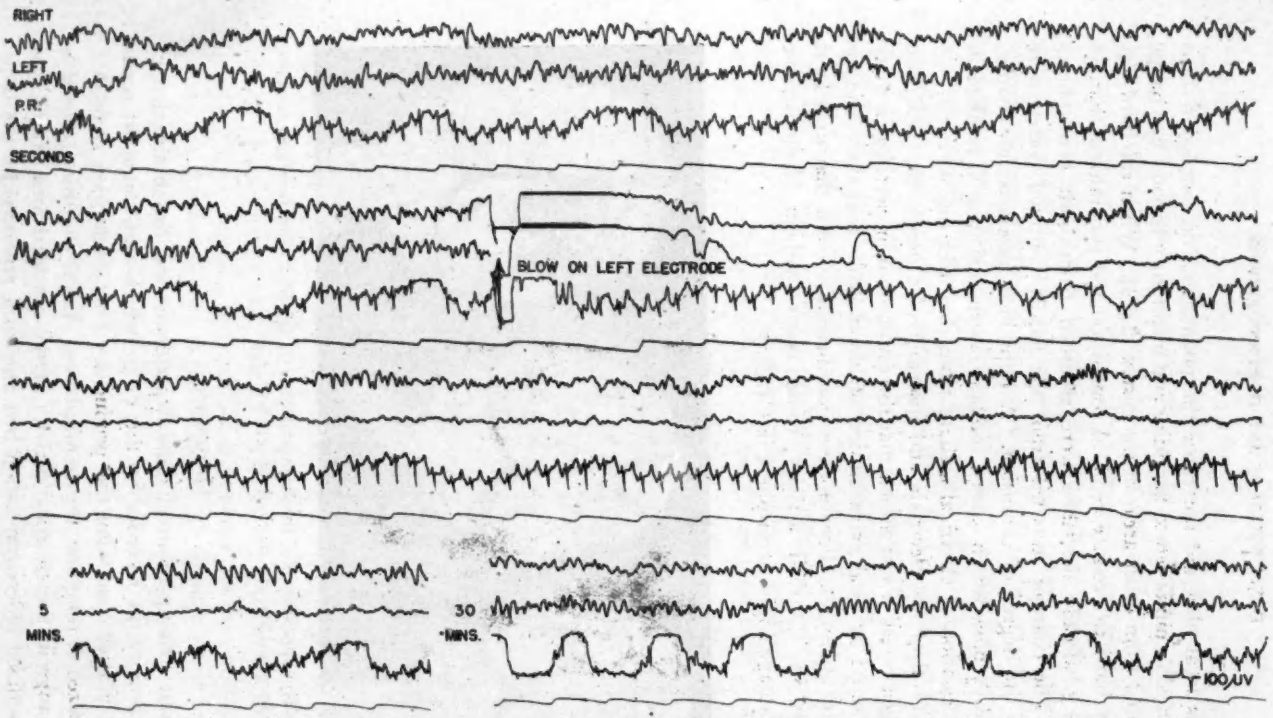


FIG. 4 (*Cat 5*).—*Localized cerebral trauma without concussion.* The recording is identical with that in fig. 2, but the screw electrode in the left parietal bone is 3 mm. longer than on the right. After a heavy blow on the left electrode there was no alteration in reflex activity. The electrical activity from the right hemisphere continued unchanged after an artefact lasting for seven seconds, but there was almost complete cessation of electrical activity recorded from the left electrode. During a five-minute interval in the fourth record the electrode was removed, cleaned and replaced, and the dura was seen to be normal. The diminution in activity was therefore not related to any mechanical defect. After an interval of thirty minutes the activity had almost returned to its former level. Delayed temporary bradycardia occurred in the absence of concussion.

When this local blow did not cause the phenomena of concussion—loss of the corneal or pinna reflex, flaccidity and inspiratory spasm—no change was observed from other areas of the cortex (fig. 4). When the blow was sufficiently hard to produce cerebral concussion the generalized changes in the e.e.g. already described were recorded from both hemispheres (Cat 5). The changes recorded from the locally contused cortex were related to the degree of contusion. A light blow produced no change, while a heavy blow which caused cerebral destruction (Cat 5) resulted in complete cessation of electrical activity from that area (fig. 4).

By removal of the electrode and insertion of a needle electrode into the damaged cortex it was shown that the diminution in electrical activity was not caused by local extravasation of blood either into the cortex or between the meninges. Post-mortem examination showed that such local diminution in electrical activity could occur without demonstrable cerebral damage (right hemisphere in Cat 5).

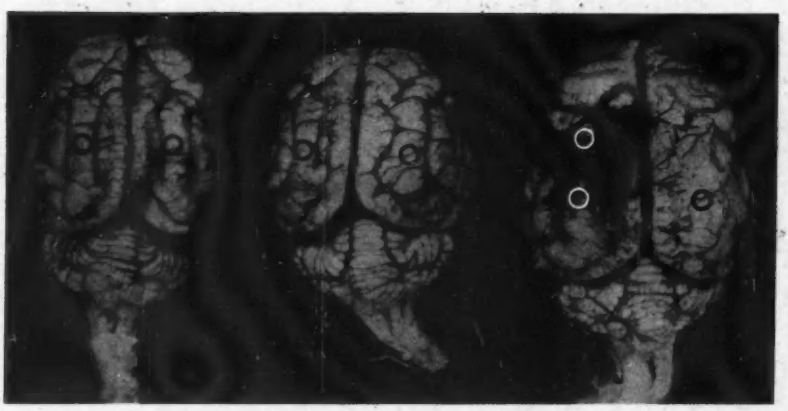

FIG. 5.—The appearance of the brains of Cats 3, 4, and 5 after formalin fixation. The electrode positions are marked by circles.

## DISCUSSION.

The most striking result of these experiments was the diminution in electrical activity, as recorded in the e.e.g., which was found to occur immediately after concussion was produced. This diminution seemed to be generalized throughout the hemispheres. Local contusion caused a similar change in the electrical activity of the contused area, which was sharply localized unless concussion had also been produced by the blow.

This flattening out of the e.e.g. record, which has been illustrated in figs. 1 to 4, resulted from a decrease in the voltage of the dominant six-a-second wave, often with complete abolition of the faster frequencies of over 14 a second. Diminution in activity of this kind may be real, as in dying cerebral cortex, or it may be only apparent when it results from loss of synchronization caused by disorganization of the pace-making mechanism. Such disorganization has been shown in animals and man (Berger, 1940, Adrian and Matthews, 1934) to be related to an increase in cerebral activity, when the retina is illuminated or the subject's attention is fixed. Disappearance of the normal frequencies seen in the e.e.g. or diminution in their voltage may also result from cerebral inactivity, in deep sleep (Loomis, Harvey and Hobart, 1935), profound anæmia (Lennox, Gibbs and Gibbs, 1939) or destruction of cerebral tissue (Martin and Williams, 1939). This state of "electrical quiescence" produced by experimental concussion undergoes a gradual change which is heralded by the appearance of slow waves of low voltage. These slow waves gradually increase in size and number and they are identical with those observed in the recovery period following concussion in man (Williams, 1941 (*a*) and (*b*)). They have been observed within fifty minutes of cerebral concussion in human subjects and they may persist for days or weeks, their duration having a direct relationship to the severity of the head injury. An e.e.g. without any evidence of such slow activity has been recorded ninety minutes after a mild head injury in a patient with concussion and an amnesia of ten minutes (Williams, 1941 (*c*)). Such a rapid disappearance of these abnormally slow waves has been seen in these present animal experiments, and it is presumed that in these instances full recovery has already occurred. In the work already referred to it was shown that during recovery from concussion in man the waves increase in frequency as they decrease in voltage and number, while the normal dominant frequencies reappear as the abnormal waves subside. It is therefore possible that the appearance of the slow waves is the first evidence of recovery of the cerebral tissue from the effects of concussion, and that the absence of or diminution in activity in the e.e.g. represents the cerebral component of the traumatic central paralysis which is shown in lower levels by complete abolition of reflex activity (Denny-Brown and Russell, 1941 (*b*)). In support of this view was the observation in Cat 2 of great diminution in electrical activity which gradually progressed to complete inactivity after a fatal blow upon the occiput, as well as by the reversible changes observed by Lennox, Gibbs and Gibbs (1939) during carotid sinus faints in man. Here the profound fall in blood-pressure with consequent cerebral ischæmia is associated

with transitory slow activity which gives place to complete inactivity during the period of unconsciousness, when the patient is flaccid and reflexes are lost. As he recovers very slow waves appear in the electro-encephalogram, and these increase in size and frequency until finally the normal dominant eight- to twelve-a-second frequency is resumed. In such a case the changes are relatively slow, but the cessation of cerebral function caused by concussion is instantaneous, so that the transition from normal electrical activity to complete abolition of this activity is telescoped and so cannot be observed. Although recovery from concussion is slower than from a carotid sinus faint, it is associated with the same stages of recovery in the electro-encephalogram as those already described.

The high voltage slow waves to which we have referred are so characteristically seen in man during traumatic unconsciousness and afterwards that it was surprising to find in animal experiments that their onset was delayed and gradual. A similar condition may exist in human subjects, for no observations have been made in man during the period of complete reflex inactivity after a head injury, and in animals reflex activity, judged by the pinna and corneal reflexes, had always returned before the abnormally slow waves appeared. Human patients remain comatose or semi-comatose long after the recovery of the reflexes, and during this time the slow waves are invariable, and their disappearance may be delayed beyond the period of complete olinical recovery although they have been found to be absent as soon as ninety minutes after concussion. As the present results suggest that the slow waves represent a stage in recovery from the abolition of electrical activity associated with traumatic paralysis, it is evident that they also support the deduction of Denny-Brown and Russell (1941) that the process of recovery from traumatic paralysis is much more rapid in the lower levels of the nervous system than in the higher.

Localized cerebral trauma, in one instance without demonstrable lesion, showed a cycle of changes in the e.e.g. closely resembling those of concussion, but limited to a very small area. When a blow caused concussion as well as local injury to the brain the changes were widespread through the hemispheres. Thus a reversible change in cerebral activity resulting from trauma may be local or general. When it is general "concussion" occurs.

The immediate onset of the changes in the e.e.g. in these experiments, seen in the instantaneous cessation of activity which occurs even before bradycardia has appeared, strongly supports the conclusion that concussion results from the direct physical effects of the blow upon the neurones

themselves. Tissue œdema (Courtney, 1899), anoxia (Trotter, 1923), hypoglycæmia (Dixon, 1940), and other secondary factors all require a certain finite time for development. Furthermore, the electrical changes are not related to cardio-respiratory changes, for they are not affected by artificial respiration or modified by the cardiac rate. Lastly, it has been shown that direct physical violence to a small group of cells and to the whole cerebral structure produces identical changes.

## Summary.

Local and general cerebral trauma has been produced in cats under light anæsthesia. Continuous electro-encephalograms were recorded throughout the experiments from points in both hemispheres by means of electrodes fixed into the skull, in contact with the dura mater. Records of cardiac and respiratory changes were also made and alteration in reflex activity and spontaneous movement were noted.

Blows resulting in concussion always caused an instantaneous and generalized diminution in the cerebral electrical activity as recorded in the electro-encephalogram, which persisted after reflex activity had returned. There was then a delayed appearance of abnormally slow waves similar to those seen after concussion in man. Reflex activity returned before these abnormally slow waves were observed.

The cerebral electrical changes caused by local trauma were limited to the injured area, but they were similar to the generalized changes seen in concussion.

No relationship was found between the cerebral electrical changes and the alterations in the cardio-respiratory mechanism or in reflex activity.

It is concluded that:

(1) Concussion is associated with diminution or cessation of the electrical activity of the whole cerebral hemispheres, as recorded in the electro-encephalogram.

(2) The delayed appearance of abnormally slow waves of cerebral electrical potential change may represent a stage in recovery from concussion.

(3) The reversible changes caused by local and general cerebral trauma are identical.

(4) Concussion is the direct result of mechanical violence to cerebral cells. It is not dependent upon secondary changes such as œdema, anoxia or hypoglycæmia for its initiation.

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# THE COMPONENT REFLEXES OF MICTURITION IN THE CAT. PART III.[1]

BY F. J. F. BARRINGTON.

*(From the Laboratories of the Surgical Unit, University College Hospital Medical School.)*

In previous papers (Barrington, 1921 and 1931) various component reflexes of micturition in the cat were described. The second of these was evoked by running water through the urethra, gave rise to a strong contraction of the bladder and was abolished by division of either the pelvic or pudic nerves, but not by division of the hypogastric nerves. Its afferent path was therefore in the pudic nerves and its efferent in the pelvic. It was abolished by transection of the spinal cord.

The second reflex can be easily demonstrated in a decerebrate, female cat having the hypogastric nerves and the bladder neck divided and a cannula tied into each cut end of the latter. The bladder cannula is connected with a graduated, warm-water reservoir which can be raised or lowered. The urethra cannula is connected with a vertical glass tube which can be filled with warm water to any required level: 3 cm. of the tube used had a capacity of 1 c.c. The bladder volume, at a given pressure, having become constant, the pressure in the urethra tube is raised 5 cm. every two minutes until the water escapes from the external meatus and the pressure in the urethra tube falls in consequence; at this moment the bladder contracts. The pressure in the urethra tube which leads to the escape of water from the external meatus in this way is found to be commonly less than 30 cm. of water and only very rarely more than 45 cm. Subsequently a strong bladder contraction can always be obtained by suddenly putting the urethra into communication with the tube when water in it is at a slightly higher pressure than that already found to cause an escape of water from the external meatus.

In making experiments in this way it was noticed that in a certain number a contraction of the bladder occurred on running water through the urethra after the pudic nerves had been cut and that in those where

[1] Parts I and II, *Brain*, 1931, **54**, 177.

such a contraction did not occur it could generally be brought about by injecting water rapidly through the urethra cannula with a syringe. The contraction was smaller than that obtained before division of the pudic nerves (fig. 1). During, and for some time after, the contraction evoked by running water through the urethra before division of the pudic nerves any pre-existing rhythmic contractions are generally (fig. 1), but not always (fig. 2), inhibited, whereas during the contraction evoked in the same way after division of the pudic nerves they are not. This contraction can be obtained after division of the spinal cord close behind the ribs, but not after division of the pelvic nerves. It can often be obtained more readily if an interval of some hours is allowed to elapse after the operation, and it seems that the division of the pudic nerves is the operative procedure which matters, since if this has been done some days previously the reflex can easily be obtained without any delay.

This reflex is abolished by division of the branches of the pelvic plexus to the urethra, whereas the second reflex is not. The branches of the pelvic plexus to the urethra can be divided and those to the bladder left intact by the following operation. The trunk of each pelvic nerve is exposed extra-peritoneally through a median suprapubic incision and then the bladder neck exposed, the cannulæ tied in and the neck divided in the usual way. The urethra is then dissected backwards off the ventral surface of the uterus for 1 to 2 cm. and the uterus divided between ligatures as far back as possible. This opens the peritoneum and leaves a leaf of parietal peritoneum on either side, to the external surface of which the branches of the pelvic plexus and certain vessels adhere; each leaf is divided in a dorsal direction between ligatures applied sufficiently far back as not to involve the more anterior branches of the plexus which can easily be seen since the pelvic nerves have already been exposed. If anything is done after this operation which produces straining the bladder may be extruded through the incision, but apart from this accident it is quite easy to obtain a large contraction of the bladder by running water through the urethra before the pudic nerves have been divided (fig. 3), but none can be obtained after.

The reflex was usually elicited by running warm water through the urethra cannula from a large reservoir, which could be raised or lowered, for measured periods, generally of thirty or sixty seconds, and measuring the volume of water which ran through. In four experiments in which the pudic nerves had been divided some days previously the minimum rates of flow per minute which produced an observable volume alteration (2½ c.c. or more) were 6 c.c. or less, between 5 and 22 c.c., between 50 and

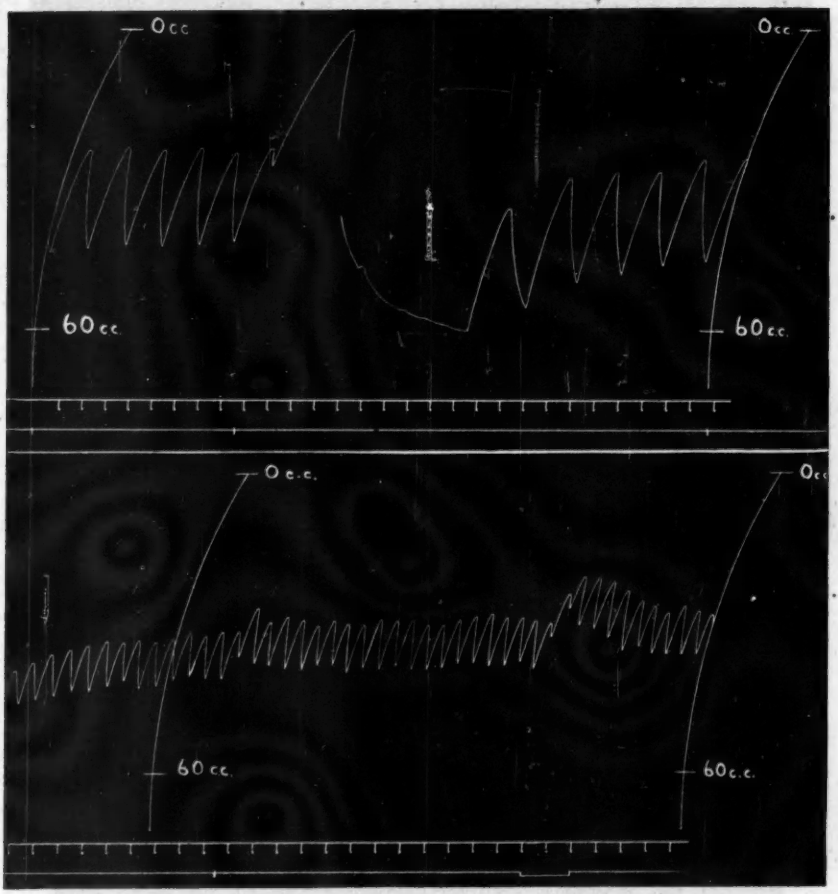


FIG. 1.—Bladder volume of a female decerebrate cat at 7½ cm. of water pressure. The hypogastric nerves and the bladder neck had been divided. At the signal in the upper part and at the first signal in the lower the cannula in the urethra was suddenly put into communication with a vertical tube of warm water, filled to 45 cm. pressure: this resulted in the escape of water from the external meatus and a fall in the water level in the tube. During the second signal in the lower part the urethra cannula was in communication with a large warm water reservoir at 75 cm. pressure for one minute and during this time 170 c.c. ran through the urethra. Twenty-six minutes elapsed between the signal in the upper part and the first in the lower. During this time, and fourteen minutes before the first signal in the lower part, the pudic nerves were divided. Time marker 30 secs.

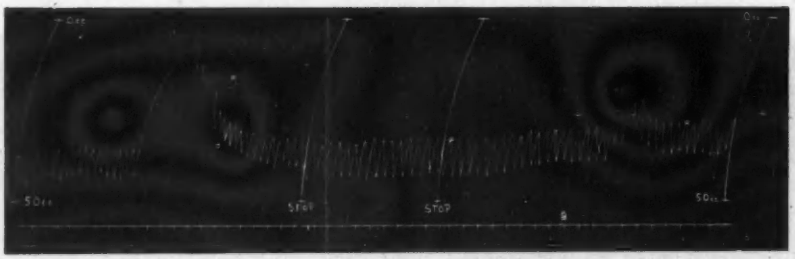

FIG. 2.—Bladder volume of a female decerebrate cat at 7½ cm. of water pressure. The hypogastric nerves and the bladder neck had been divided. At the first and second signals the urethra was put into communication with a vertical tube of warm water filled to 45 cm. pressure: the water escaped from the external meatus at once in each case and the pressure in the tube fell. During the third signal water was run through the urethra from a large reservoir at 60 cm. pressure for one minute: 48 c.c. ran through in the minute. The pudic nerves were divided during the first stop. Time marker 30 secs.

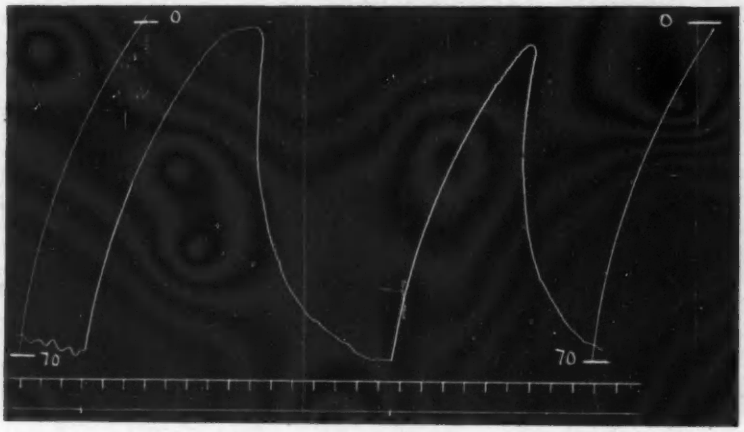


FIG. 3.—Bladder volume of a female decerebrate cat at 7½ cm. of water pressure. The bladder neck, hypogastric nerves and branches of the pelvic plexus to the urethra had been divided. At each of the two signals a vertical tube of warm water at 30 cm. pressure was suddenly put in communication with the urethra and water escaped from the external meatus at onec. Time marker 30 secs.

100 c.c., and between 100 and 160 c.c. respectively. The mean rate of flow from the vertical tube which was used in experiments to elicit the second reflex was found on two experiments to be 17 c.c. per minute and 25 c.c. per minute respectively for shorter periods than thirty seconds. As these figures are intermediate between the extremes of the previous four it

explains why the reflex may at times be elicited by this method and not at others.

In six adult female cats, believed to be normal, the time taken by a single act of micturition and the volume of urine passed were measured once in each. The volumes passed varied from 88 to 202 c.c. The mean rate at which urine was passed varied from 203 to 288 c.c. per minute and averaged 253 c.c. per minute. The mean rate of flow of urine at a normal micturition is therefore much greater than that necessary to evoke the reflex in a decerebrate cat under the conditions of the experiments.

Running water through the urethra can therefore produce a bladder contraction through the action of at least two reflexes, firstly through reflex two, which is a hind-brain reflex having its afferent path in the pudic and efferent in the pelvic nerves, and secondly through a spinal reflex (reflex seven) which has both paths in the pelvic nerves; the former is more easily elicited and gives rise to a greater contraction.

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# VISUAL DISORIENTATION WITH SPECIAL REFERENCE TO LESIONS OF THE RIGHT CEREBRAL HEMISPHERE.

BY W. RUSSELL BRAIN.

*(From the Neurological Department, London Hospital.)*

THE visual perception of the spatial relations of objects in the external world is an extremely complex function which has interested both experimental psychologists and clinical neurologists for many years. The contribution of clinical neurology to its elucidation has been the attempt to interpret the symptoms of spatial disorientation and to correlate them with focal lesions of the nervous system. This task has proved difficult for several reasons. Visual disorientation is not a very common symptom of a focal lesion of the brain and the number of cases studied has been small. In many of these the lesions being caused by arteriosclerosis have been multiple and situated in both cerebral hemispheres. Even traumatic lesions are apt to produce widespread injury to the brain, and in many cases of visual disorientation caused by gunshot wounds in the last war both cerebral hemispheres were extensively damaged. A more formidable difficulty is the complexity of the symptoms usually produced by any lesion large enough to cause symptoms at all.

Broadly speaking, until the last war visual disorientation was regarded mainly as a manifestation of agnosia occurring in patients with other perceptual disorders of that kind. The investigation of cases of gunshot wounds of the brain during the war, however, showed that visual disorientation might occur without object-agnosia and its manifestations were thus studied in isolation, especially by British neurologists. Among the earlier reported cases may be mentioned those of Hughlings Jackson (1876), Wilbrand (1892) and Wendenburg (1909); and von Stauffenberg (1914) in his review of mind-blindness discusses spatial disorientation in that connection.

Riddoch (1917) in his paper on the effects of occipital injuries reported a case, No. 3 in his series, of a patient who was unable to localize an object seen, to tell which of two objects was the nearer to him and to appreciate visually depth and thickness in objects, notwithstanding perfect consciousness of light and shade and possession of binocular vision.

Holmes (1918) reported a group of six cases of disturbance of visual orientation, and later (1919) summarized the abnormalities observed in eight cases. Though the clinical picture differed somewhat in different cases, the cardinal features of the patients in this group were as follows: (1) There was a disorder of absolute localization of objects seen, leading to an inability to touch them or indicate their position correctly. (2) Relative localization was also impaired so that the patient was unable to say which was the nearer of two objects. (3) The patient was unable to determine the relative lengths of two lines or the relative sizes of two objects. (4) Walking revealed a gross disorder in spatial orientation. Patients would run into objects which they saw clearly and have great difficulty in finding their way about and in learning the topography of the room. For example, a patient intending to go to his bed would see where it was but set out in the wrong direction. In some cases topographical memory for experiences which occurred before the injury was defective. One patient was unable to describe routes previously familiar to him. Memory for objects, however, was preserved. (5) There were difficulties in picking out objects visually and in counting them, and also in reading. (6) Recognition of movement in a sagittal plane, i.e. to and fro, was defective, but lateral movement was recognized. (7) Visual attention was impaired. (8) Various disorders of the ocular movements were observed. (9) In one case only was stereoscopic vision lost. In all Holmes' six cases both cerebral hemispheres were injured. He stated that he had not seen similar symptoms produced by unilateral lesions and that "it may be assumed that bilateral lesions are necessary for their appearance." The lesions responsible for the symptoms were localized in the posterior and upper parts of both parietal lobes. In the two cases in which post-mortem examinations were obtained the lateral lesions involved the angular and supramarginal gyri, while the mesial were placed immediately dorsal or posterior to the splenium. Holmes and Horrax (1919) reported a case in which in addition to visual disorientation there was loss of stereoscopic vision.

Kleist (1922) in his review of war injuries of the brain distinguishes the following forms of visual disorientation. (1) Defective relative localization of objects in the visual field may occur as a result of lesions of the primary visual paths and is therefore associated with visual field defects. (2) Disturbance of absolute visual localization (Ortsblindheit) is independent of visual field changes. It leads to "optical ataxia," misdirected grasping and pointing, though these acts are still carried out correctly in response to tactile stimuli. Kleist distinguishes the disturb-

ance of absolute visual localization from (3) agnosias for visual space (Optisch-raümliche Agnosien, Orientierrungsstörungen). These terms, he holds, should be reserved for cases in which there is a loss of visual memories. This is found in two disorders (i) optic-somatic disorientation and (ii) spatial memory loss (Ortsgedachtnisverlust), which is attributed to a loss of engrams of spatial sense. He compares the difference between disturbance of absolute localization and loss of spatial memories with that between perceptual form-blindness and visual object-agnosia. Finally he notes that agnosia for visual space must be distinguished from visual disorientation secondary to visual object-agnosia.

Pötzl (1928) groups together as the spatial forms of agnosia, cerebral metamorphopsia, psychical paralysis of gaze and the visual disorders of spatial orientation. As an example of the last of these he reports a case of visual disorientation resulting from a lesion of the right cerebral hemisphere, which, together with his analysis of it, is discussed below. Riddoch (1935) reported two cases in which visual disorientation was limited to homonymous half-fields, in both cases on the right side. Two recent reviews of agnosia by Quensel (1931) and Lange (1936) include discussions of visual disorientation, but add little to the subject.

The cases reported in this paper fall into two groups, which will be described and discussed separately. Group 1 comprises three cases in which there was defective visual localization of objects in space. In all three the lesion was unilateral, being on the right side in two and the left in one, and the visual disorientation was limited to the opposite homonymous half-fields. In Group 2 there were three patients who showed a more profound disturbance, characterized by an inability to follow familiar routes owing to a tendency to take turnings to the right instead of to the left. In all three the lesion was a massive one in the right parietal lobe, and the disorder of route finding is ascribed to an inattention to, or neglect of, the left half of external space. In conclusion, the relationship between visual disorientation and awareness of the body, and the nature and classification of visual disorientation are discussed.

## Visual Disorientation Limited to Homonymous Half-fields.

Three varieties of visual disorientation limited to homonymous half-fields have been described: (1) When vision in homonymous half-fields is impaired but not completely lost, objects situated within the amblyopic half-fields may be vaguely seen but incorrectly localized (Kleist, 1922; Poppelreuter, 1923). This defect, which is of the same kind as impairment of visual discrimination of two points and of the form of objects in the

same circumstances, is the result of a lesion of the primary visual pathway between the retinæ and the visual cortex. (2) Fuchs (1920) points out that in homonymous hemi-amblyopia objects presented to the amblyopic fields may appear to be displaced towards the fixation point. This is due to a displacement of the median plane. A new functional centre arises in the seeing half-fields and the old fixation point is displaced towards the blind side. The localization of an object seen in the amblyopic fields relative to the old fixation point is transferred to the new centre and the object is thus apparently displaced towards the seeing side. (3) Finally visual disorientation may occur in homonymous half-fields without any limitation of the visual fields for ordinary stimuli or serious disturbance of central vision (Riddoch, 1935). Only the third of these disorders is the result of a disturbance of the central integration of visual space perception, and it is to this group that the following three cases belong.

*Case 1.*—H. L. L., right-handed female, aged 36. Compound fracture of skull: fragments of bone driven into left parietal lobe removed at operation. Aphasia, agraphia with tendency to mirror-writing, acalculia, visual disorientation in right homonymous half-fields, impaired discrimination of right and left, dysæsthesia of right half of body, allæsthesia for movement, apraxia for dressing.

*History.*—On October 16, 1940, she was admitted to hospital as an air-raid casualty, unconscious, with a penetrating wound in the left posterior parietal region. The scalp edges were excised, the wound in the skull was enlarged to ¾ in. and fragments of bone were removed from the brain which contained a hole about 1½ in. deep. She regained consciousness after operation at 11 a.m., her last memory being of going to bed at 11 p.m. the previous night. She complained of loss of smell. A marked herniation of the brain developed, but later shrank back into the skull. X-rays showed fragments of bone remaining in the brain (figs. 1 and 2).

On November 20, 1940, Mr. D. W. C. Northfield skin-grafted the fungus, and on December 20 she was discharged from hospital and later readmitted. On January 16, 1941, Mr. Northfield operated and removed the five bone fragments from the brain. The injured area of the brain was about 2 cm. across and was situated in the superior parietal lobule close to, if not incorporating, the upper end of the sensory cortex. A cylinder of brain including the bone fragments was enucleated, and the resultant cavity passed medially and exposed the falx but did not reach the corpus callosum.

Operation was followed immediately by marked aphasia, weakness of the right lower face and right upper limb and some sensory loss of the cortical type in the right hand. These symptoms disappeared within a month, but on February 17, 1941, when she was up, she complained that she ran into objects on her right side and she was found to exhibit visual disorientation in the right half-fields. She experienced difficulty in recognizing her own right and left sides and right and left in her environment, but she said that this symptom had been present since her accident, and was not worse since her last operation.

On February 24, 1941, she described her symptoms as follows:

*Visual orientation.*—She always knew where she was and on getting up had no difficulty in finding her way about, though she had been a long time in bed. When

FIG. 1.

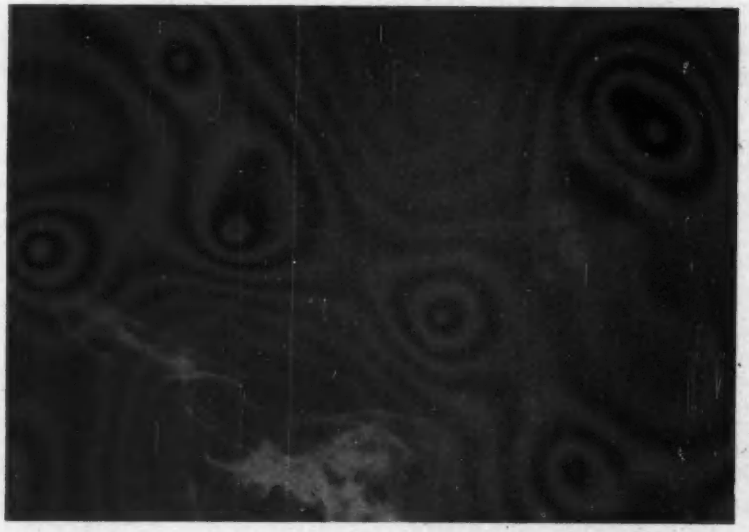

FIG. 2.

FIGS. 1 and 2 (*Case I*).—Radiograms of skull showing position of bone fragments in left parietal lobe.

she got up she found she tended to run into objects on her right side and still did so, because she did not know how near they were. She said: "I couldn't feel them until I actually hit them." A nurse had to escort to prevent her from knocking herself against things. If she ran into an object she had no difficulty in finding her way round it. She had no difficulty in finding her way about when she returned home, and she had no loss of memory for places. She experienced a similar difficulty in finding objects within reach of her hand on the right side, e.g. a cup, because she could not judge its distance accurately.

*Awareness of body.*—She said "All the right side feels as if it doesn't belong to me. When I move my right hand I don't feel I'm moving my hand myself, although it goes." She was apt to lose her right slipper without knowing it.

*Awareness of right and left.*—Since the injury she had found it very difficult to distinguish her right and left sides; she could only identify her hands through the knowledge that her wedding ring was on her left hand.

*Dressing.*—She had great difficulty in putting on her clothes. She attributed this to the fact that she did not know the right side from the left. She might put them on back to front or upside down.

*Writing.*—At first after her injury she could not write at all. The ability to do so gradually returned, but when she wrote with the right hand it felt as though she was writing with the left. At first she tended to write in mirror fashion, the letters being reversed or upside down, e.g. "u" instead of "n".

*Calculation.*—She was unable to do sums or reckon up the cost of a series of articles or the change due from a purchase. When buying things in hospital she had to leave this to other people.

*Examination.*—Intelligent and co-operative. No disturbance of utterance or comprehension of spoken speech.

*Visual functions.*—The optic discs were normal. Visual acuity was: right J2 and left J1 (corrected). The visual fields were full to 2/1500, 10/1500 and 10/330 white; there was no visual inattention. She read accurately but said that after reading for a time she became tired and print appeared misty. In writing the words "Chase Farm Hospital" she made several mistakes which she corrected. She recognized and correctly named common objects and used them correctly. She was able to reproduce patterns made with matches promptly and accurately, and to copy line drawings. Objects seen in the right half-fields were inaccurately localized. Thus when asked to fix centrally and touch the observer's finger held in her right half-fields she missed it because she deviated to the left and towards the horizontal axis. When allowed to look at an object held towards her right side and then to point to it with her eyes closed she made a similar mistake. When allowed to look at an object held to her right side and asked to point to it she pointed to the right of it. All the errors were of a range of a few inches. Localization in the left half-fields was normal. In the ward she walked rather slowly and avoided a stool placed in her path, giving it a wide berth. She found her way back to her own bed, but on two occasions when halted some yards from her bed and asked to point to it with eyes closed she pointed to a bed on the right of it. She was able to find the middle point of a line and the centre of a circle without difficulty. She recognized which of two lines was the longer, and with some hesitation which was the nearer, but she was slow in understanding the meaning of "nearer" and "farther."

*Discrimination of right and left.*—When asked to indicate the right and left sides either of herself or the observer she was slow and inaccurate. She first called her right hand left and vice versa. She then correctly named the hands of the observer who was facing her. She next got her own hands right; and then hesi-

tated over the observer's but named them correctly. When the observer turned round with his back towards her she was very slow but correct. She named her own right and left eyes correctly; recognized her own fingers, moved individual fingers on command and imitated the observer's finger movements correctly.

The pupillary reactions were normal and there was no defect of ocular movements on command or on following a moving object in any direction.

Apart from anosmia in both nostrils the remaining cranial nerves were normal.

There was slight weakness of movements of the right upper and lower limbs, and the tendon reflexes on the right side were slightly exaggerated. The plantar reflexes were flexor; no cutaneous anæsthesia or impairment of tactile localization or discrimination; no analgesia. Replies to tests of appreciation of passive movement of the fingers and toes were slightly slower on the right side than on the left, but accurate.

*Case* 2.—S. B., right-handed male, aged 29, lorry driver. Meningioma of convexity of right parietal lobe; focal convulsions; defective memory for routes. Post-operative visual disorientation in left homonymous half-fields, dysæsthesia of left half of body, allæsthesia for movement.

*History.*—Eleven years ago he began to have fits which were sometimes precipitated by distension of the bladder or rectum. The aura consisted of palpitation and a peculiar feeling spreading up the left upper limb to the left side of the neck and followed by flexion of the left elbow. The aura usually lasted about two minutes and the attack could often be aborted by tightening a belt round the arm above the elbow. Failing this, consciousness was lost for about two minutes. Information about the phase of convulsion was scanty, but he was said to foam at the mouth occasionally, but not to bite his tongue or wet himself. There was no post-epileptic paralysis; headaches usually occurred after a fit, but not at other times. The attacks occurred at night only for many years, at irregular intervals of from eighteen months to a fortnight. Recently they had occurred by day also, and more frequently.

He also complained of constant slight weakness of the left upper and lower limbs; and occasionally there was transitory loss of power in the upper limb lasting for half an hour. At times the left great toe felt as if it was not there. His memory was poor, especially for routes. He noticed in the course of his work that if he was driven over a route he could not find it again by himself, because he could not remember the turnings: his memory for places was good.

*Examination, June 5, 1941.*—Intelligent and co-operative. No aphasia, apraxia or object-agnosia. Normal discrimination of right and left. Visual acuity: R. and L. $\frac{6}{6}$; fields full to 10/330, 10/1500 and 2/1500. No visual inattention. Cranial nerves including optic discs normal. Right upper and lower limbs normal. Slight hypotonia and very slight weakness of left upper and lower limbs. Reflexes: arm-jerks—left brisker than right; abdominal reflexes sluggish but equal; knee-jerks—left brisker than right; ankle-jerks equal; plantar reflexes flexor. Sensation: no anæsthesia to light touch, no impairment of tactile localization or appreciation of vibration. No analgesia to pin-prick on any part of the body; postural sensibility normal in right upper and lower limbs and left upper limb; grossly impaired in left great toe; tactile discrimination normal on right side and fingers of left hand; no threshold obtainable on left foot. Figures drawn on left shin were unrecognized.

X-rays of skull (figs. 3 and 4) showed scattered areas of calcification in the brain and increased vascularity of the skull in the upper part of the right parietal lobe.

*Operation.*—On June 26, 1941, Mr. D. G. Phillips operating through a right occipital flap exposed and removed a meningioma of the right convexity. The tumour which was bun-shaped and measured 5 to 6 cm. across compressed the right

FIG. 3.

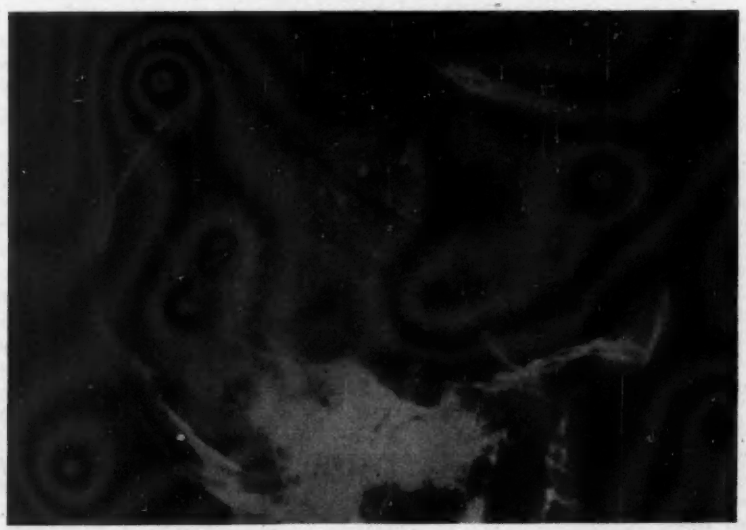

FIG. 4.

FIGS. 3 and 4 (*Case* 2).—Showing calcification in meningioma of right parietal lobe and abnormal vascularity of overlying skull.

parietal lobe, approaching the falx medially, but not impinging upon it, and its anterior border impinged upon the upper third of the post-central convolution. During the removal of the tumour there was much swelling of the brain so that by the time the tumour had been completely removed there was only a faint saucer-shaped depression in the brain. On recovering consciousness he showed a complete flaccid left hemiplegia. During the first three days after operation he was excitable and resistive, but he then became mentally normal. The hemiplegia started to improve three days after the operation when he became capable of slight movement of the left upper limb.

*Examination, July 10, 1941.*—Normally orientated, co-operative, good power of attention. No aphasia, no loss of topographical memory.

*Appreciation of absolute and relative distance.*—Since the operation he has had difficulty in judging distances on his left side. A person walking towards him along the ward on the left at first seems a long way away and then "suddenly they are on top of me." "People coming from that side seem to glide rather than walk, they move so fast," but he had no difficulty of this kind to his right side. He added that sometimes people walking down the ward from the left seemed to walk right through the table. When asked to judge the relative distances of objects in the ward he made gross mistakes with objects on his left side. For example, he said a patient in bed on the opposite side of the ward about 18 ft. from him was nearer than a vase of flowers on a table about 8 ft. away. He was always correct, however, in judging relative distances on his right side. When tested with small objects held at varying distances within about a yard from his face he was always quite accurate in saying which of two objects was the nearer to him on either side. When asked to point to an object held in the left half of his visual field either while his eyes were open, that is in a position of central fixation, or after a previous glance at it with his eyes closed he always made errors of a few inches which did not appear to be consistently in any single direction. In this test he was always quite accurate with objects presented in the right half of his visual field. He was able correctly to divide a line, to indicate the centre of a circle and to show the relative length of three lines.

*Visual recognition of objects.*—He was able to recognize promptly a series of common objects shown him. In order to test whether he had visual object-agnosia limited to one-half of the visual field, common objects were shown him slightly to one or other side of the central fixation point. He either recognized the objects or correctly described their shape and was as successful with objects presented to the left of the central point as to the right.

*Reading and writing.*—He read correctly a passage from a newspaper and showed that he understood its meaning. In writing he tended to omit letters and he said that writing felt more difficult than before his operation. There was no mirror-writing. He wrote his name and "God save the King" correctly, and "Chase Farm Hospital" with slight errors in the formation of letters.

*Calculation.*—In successively subtracting 7, starting from 100, a pre-operation test gave the following result: 100, 93, 86, 79, 72, 67, 60, 53, 46, 39, 32, 27, 20, 13, 6. Two weeks after operation his response was slow and hesitating and the test yielded the following result: 100, 93, 69, 62, 67, 60, 57, 50, 43, but he was able to state the correct change in purchasing a 1s. 6d. object with 10s. and 1d. object with 2s. 6d.

*Use of objects and construction.*—He was able to use objects correctly with his right hand, and to copy patterns constructed with matches.

*Awareness of body.*—He said that the left side of his body below the neck sometimes felt as if it did not belong to him. When his left hand was lying in

bed it might feel as if it were against his face. This feeling was so real that although he could see where his left hand was he had to touch it with his right hand to verify its position. Sometimes when using his right hand it felt as though he were using the left or vice versa. He was fully aware of the weakness of his left upper and lower limbs. He was able to recognize and name his fingers, and to imitate finger movements with his right hand; his left hand being too weak.

*Discrimination of right and left.*—He knew his own right and left sides; in naming those of the observer he was slow, but accurate.

*Physical examination.*—Optic discs normal. Visual fields full on confrontation: no visual inattention. Pupillary reactions normal. Ocular movements full; slight nystagmus on lateral fixation to right and left. Slight analgesia on left side of face. Some weakness of voluntary movement of left side of face. Tongue deviated to left on protrusion. Hearing good. Sounds correctly localized on either side. Weakness and hypotonia of left upper and lower limbs. Severe loss of postural sensibility in fingers and toes on left side, the two ulnar fingers being least affected. No threshold for tactile discrimination of left upper or lower limbs. Appreciation and localization of light touch relatively little impaired on left side of body. Touches on left hand were fairly accurately localized, but if the left upper limb was passively moved when the patient's eyes were closed localization of light touches on the left hand was temporarily impaired until he was allowed to see the new position of the limb. Slight relative analgesia in left upper and lower limbs; tendon reflexes somewhat diminished on left side. Plantar reflexes: right flexor, left no response.

*Subsequent history up to July 24, 1941.*—Improvement was rapid and by July 17 he had lost his difficulty in judging distances to the left and no longer had any abnormal feelings referred to the left half of the body. He first got up on July 20, and then experienced no difficulty in orientating himself, never got lost in the ward or in going outside to the lavatory and never ran into things. He had not dressed beyond putting on a dressing-gown and slippers but he had no difficulty in doing this.

*Examination, July 24, 1941.*—Speech, reading and writing normal. Visual fields full: no visual inattention, no errors of direction in pointing to objects in either half-fields. Left hypotonia, hemiparesis and hemiataxia. Tendon reflexes brisker on left side than right. Plantar reflexes: right flexor, left no response. Sensation: left upper limb: no cutaneous anæsthesia or analgesia, slight loss of postural sensibility in the fingers, tactile discrimination impaired in little finger only; left lower limb: some cutaneous anæsthesia and analgesia, gross loss of postural sensibility in toes, no threshold for tactile discrimination in foot, vibration sense lost.

*Case 3.*—J. K. P., right-handed male, aged 46, accountant. Glioblastoma of right temporal lobe verified at operation. Initial partial left homonymous hemianopia which disappeared spontaneously. Defective localization of objects in left homonymous half-fields. Post-operative field defect in left lower homonymous quadrants. Later complete left homonymous hemianopia owing to recurrence of tumour. Impairment of tactile discrimination in left upper and lower limbs; later impairment of postural sensibility in left hand, and left hemiplegia. Terminal complete visual disorientation for place.

*History.*—In November, 1934, he noticed luminous hallucinations described as "zigzags," a shimmering in the right half of the visual fields, followed a month later by headaches. In February, 1935, he was examined by Dr. A. Tuyl, of Valparaiso, who found bilateral papilloedema of 2-3D and left hemianopia, almost complete in the upper quadrants but sparing the macula and the mesial one-fifth of the lower quadrants. One month later Dr. Tuyl found that the visual

defect was only relative over all but the extreme periphery of the left half-fields. During the voyage to England in March the patient noticed some difficulty in judging distances to the left side. When pouring out tea, holding the teapot in his left hand, he intended to put the pot back on a table on his left, but missed the table and put the pot on the edge. His only other symptoms were giddiness on one occasion, slight general unsteadiness and a tendency to be nervous about himself.

*Examination, April 17, 1935.*—No mental abnormality. No aphasia or apraxia. Slight bilateral papilloedema, not more than 1D in right eye and less in left. Visual acuity $\frac{6}{6}$ in both eyes. Visual fields full to 9·5/2000. Central vision normal. Asked to look at objects in various parts of the visual fields and then point to them with eyes closed he was accurate with objects in the right half-fields but inaccurate to the left, pointing about 3 in. to the left of the true direction. Pupillary reactions normal. Ocular movements full; fine nystagmus on lateral fixation, more marked to right than to left. Slight weakness of left lower face. Left upper limb: slight hypotonia and slight ataxia in finger-nose test. No muscular weakness in limbs. No sensory loss to light touch, pin-prick, postural sensibility or tactile discrimination.

It was observed that on several occasions when he was trying to shake the ash off his cigarette into an ash-tray on his left side he missed the ash-tray and dropped the ash on the floor.

On May 7, 1935, a defect of tactile discrimination was observed in the three ulnar fingers of the left hand and the sole of the left foot, and responses to passive movements of fingers of the left hand were slower and less certain than on the right side.

*Operation.*—On May 9, 1935, Professor Hugh Cairns removed a large tumour of the right temporal lobe which proved to be a glioblastoma. The tumour, which weighed 50 grm., measured about 7 cm. in its antero-posterior diameter and extended backwards to within about 2 cm. of the lateral sinus. It was attached to the dura laterally over an area about 4 cm. in length and 3 cm. from above down. Its transverse and vertical diameters were about 5 cm.

*Subsequent history.*—After operation there was a left homonymous inferior quadrantic visual field defect. An unexpected sequel of operation was a motor amusia. The patient said spontaneously that he had almost completely lost his sense of tune. Previously he could go to a dance and whistle all the tunes played by the band the next morning. He now found it difficult to whistle the most familiar tunes, although he could recognize them perfectly when he heard them. Asked to whistle "It's a long way to Tipperary," he whistled something quite different and said "No, that's not it." When given the first bar or two he whistled the first two lines accurately, changing key twice owing to his inability to strike the correct intervals between the notes. His general condition improved and he remained free from headaches for five months. Symptoms of increased intracranial pressure then recurred, and were only temporarily relieved by X-ray irradiation of the tumour. His visual field defect extended to the whole of the left homonymous half-fields. During the last two months of his life he developed pains in the left palm and left lower limb which were attributed to involvement of the right optic thalamus, and during the last two or three weeks there was a progressive left hemiplegia. During this period he was often completely disorientated, thinking, for example, that he was on a liner at sea, yet he never failed to recognize anyone whom he knew. He died on March 28, 1936.

In the cases reported above the visual disability consisted in an inability accurately to localize objects seen in the crossed homonymous half-fields, in spite of the absence of any defect of visual acuity or attention. All three made errors in pointing to objects within reach of their hands when presented to them in the affected half-fields, and Nos. 2 and 3 spontaneously noticed their errors in trying to pick up objects. Nos. 1 and 2 experienced similar difficulties in localizing objects at a greater distance. No. 1 tended to run into objects on her right side because she could not judge their distance. No. 2 failed to estimate the relative and absolute distance of objects in the ward in his left half-fields after his operation, but he had lost this symptom when he got up. His inability to localize distant objects was in striking contrast with his uniform success in judging the relative distance of two objects held within a yard of him. No. 3 did not at any time run into objects; his defective localization appeared to be limited to objects within arm's length. These differences may be explained by differences in the situation of the lesion in the three cases. In Nos. 1 and 2 this was in the upper part of the parietal lobe, in No. 3 it arose in the posterior part of the temporal lobe. It seems likely that, although external space is finally presented to consciousness as one, estimation of "walking-distance" depends upon neural links between the visual cortex and the leg area of the post-central convolution, while estimation of "grasping-distance" depends in a similar way upon association paths between the visual cortex and the hand and arm area. In Nos. 1 and 2 the lesion would be likely to damage the former, and in No. 3 the latter, and before operation in No. 2 sensory loss of the cortical type was more severe in the foot, in No. 3 in the hand. This distinction between modes of exploring space finds support in the perceptual experiences of the congenitally blind who must consciously employ modes of perception which in the seeing are so subordinated to vision that they never reach consciousness. Villey (1924), himself blind from earliest childhood, distinguishes "manual space," capable of being explored with one or both hands, and "brachial space," which is investigated by arm movements; while greater distances are estimated by "the movements which the blind man makes with his legs to reach the object. The number of steps informs him as to the distance which separates him from it, the orientation of those steps as to the direction in which it is situated. With the distance and the direction he has the two indications which are necessary and sufficient to localize an object in relation to himself." In the seeing, these factors are so fused with the purely visual spatial data in the perception of space as to be unconscious. But the visual localization of an object within the reach

of the hand determines the precise combination of movements necessary to grasp it. Hence it is to be expected that the nervous organization of what is subjectively brachial space or grasp space and walking space should be partially different, and that the two should be differently affected according to whether a lesion is nearer the hand or the leg area.

As in Holmes' (1918) patients the defective localization was much more evident in the sagittal than in the coronal plane. Holmes and others have drawn attention to the difference between the visual localization of objects in the coronal plane (up and down, right and left) and in the sagittal plane (to and fro). The retinæ may be regarded as surfaces extended in the coronal plane, for their curvature in the sagittal plane has no bearing upon their rôle in the localization of visual images. A point in the retina possesses local sign, and the discrimination between two separate points of the retina simultaneously stimulated is the basis of the relative localization in the coronal plane of the two objects corresponding to the stimuli. This function has been compared with the discrimination of two points on the surface of the body simultaneously touched. The relative positions of points stimulated on the retina in itself yields no information as to the relative positions of the two corresponding objects in the sagittal plane. This knowledge is acquired by means of a further complex process. It is unnecessary here to discuss in detail the various factors concerned in the visual perception of distance. It will be sufficient to say that the disparity of retinal images in the two eyes seems to be the most important single factor in causing awareness of non-equidistance of the corresponding objects, but that definite impressions of " nearer " and " farther " and the accurate perception of absolute and relative distance require the addition of other perceptual and even ideational data, such as proprioceptor impulses from the ciliary and extra-ocular muscles, visual sensations of parallactic movement, relative sizes of retinal images, relative brightness, perspective effects and previous knowledge of the spatial arrangement of the field (Vernon, 1937). All the visual sense-data have further to be integrated with impulses from all the somatic proprioceptors which convey awareness of the position of the body in space.

Thus the relative and absolute localization of objects in the coronal plane is founded upon relationships already established at the level of the primary visual cortex, whereas localization in the sagittal plane is the outcome of functions integrated at a higher level. Nevertheless these higher functions are necessary for any accurate localization in the coronal plane also. Two images may lie close together in the coronal plane of

the retina, but the corresponding objects, if they are very distant, may be a long way apart. Only when their distance in a sagittal plane is known can the distance between them in the coronal plane be appreciated.

These facts explain why, when visual localization is defective, appreciation of relative and absolute distance and movement in the sagittal plane is more affected than in the coronal plane. A disordered perception of movement is a result of disordered perception of the spatial framework in which movement occurs and to which it is referred, for our awareness of the rate of movement of a moving body implies knowledge of the distance through which it moves in a given time. Thus No. 2 observed that people coming towards him from the left appeared to move so fast that they seemed to glide rather than walk. Impairment of appreciation of the relative distance of objects may lead to two objects appearing to be in the same place at the same time. To this patient people walking down the ward seemed to walk through the table just as to Riddoch's (1917) patient No. 3 two vehicles approaching one another in the street seemed about to collide.

In the three patients reported, the defect of visual localization was limited to the crossed half-fields; in Case 1 on the right and in Cases 2 and 3 on the left side. It follows that the central process through which objects are localized visually is organized in each hemisphere independently in respect of the crossed half-fields and is not related to the dominance of one hemisphere; a fact which has been demonstrated in another way by Akelaitis (1941), who showed that visual localization was not impaired in either half-field by complete section of the corpus callosum.

### Defective Right and Left Discrimination in Route-finding.

The following three patients exhibited quite a different form of visual disorientation. Though not suffering from a loss of topographical memory or an inability to describe familiar routes, they nevertheless got lost in going from one room to another in their own homes, always making the same error of choosing a right turning instead of a left, or a door on the right instead of one on the left. In each case there was a massive lesion in the right parieto-occipital region, and it is suggested that this, in addition to causing a left hemianopia, resulted in an inattention to or neglect of the left half of external space.

*Case 4.*—C. R. C. S., right-handed male, aged 47, glioblastoma multiforme of right parieto-occipital region verified at operation. Difficulty in finding way about, turning to right instead of to the left. Difficulty in putting on clothes. Dysæsthesia of left side of body with spontaneous allæsthesia. Left homonymous hemianopia,

spastic left hemiplegia. Anæsthesia to light touch, analgesia to pin-prick, gross postural loss in fingers and toes and loss of tactile discrimination on left half of body.

*History.*—In August, 1939, he began to bump into things on the left side. His left hand became clumsy and he began to suffer from headaches. His wife noticed that his temperament had changed and that he had become irritable. During air-raid warnings he got lost in his own room, tried to put his dressing-gown on upside down and was unable to knot his tie. He then had left homonymous hemianopia, over-action to pin-prick over the left upper limb and gross impairment of postural sensibility in the left fingers and toes. On September 30, 1939, he was operated upon by Mr. P. B. Ascroft who exposed a glioblastoma multiforme in the right occipito-parietal regions, the section of which showed large numbers of fibrillary astrocytes and also areas of less differentiated cells. He improved remarkably after operation and had a course of deep X-ray therapy from Dr. G. Cranston Fairchild, being treated with a total tumour dose of 5448 r.

In February, 1940, while on holiday he had a convulsion which began with a sensation of numbness in the left hand. The whole of the left side of the body twitched for at least an hour, but the convulsion did not become generalized and there was no loss of consciousness. He was put on phenobarbital and no further convulsion occurred. During the early summer there was increasing weakness of the left upper and lower limbs and vision to the left side deteriorated again. Headache, of which he had never been completely free, increased in severity. There was occasional vomiting and hiccup was troublesome.

*Disorders of orientation.*—During the few weeks before examination he had experienced difficulty in finding his way about. This occurred in daylight, but was worse in the dark. In going from his bedroom on the first floor downstairs to the sitting-room he tended to turn to the right instead of to the left and in consequence would arrive in the dining-room. He then recognized his mistake and this caused him irritation. He experienced no difficulty in visualizing the correct route. He found it difficult also to find the electric light switch above his head in the dark, although he could picture where it ought to be. He had great difficulty in putting on his clothes. He said that it was quite hopeless for him to try and put them on in an automatic fashion. He could put on his pyjama coat only if he laid it out first on the bed. He had learned that the handkerchief pocket was on the left side and the buttons on the right side and by utilizing these landmarks he was able to put it on. If he tried otherwise he got in a complete muddle.

*Awareness on the left side of the body.*—He complained that he did not know where the left side of his body was. He might awaken lying on his left arm and not know where it was and when eating he would hold the fork up in the air without knowing it. He said that the left side of his body felt different from the right side and as if it were on the right side. This was a very peculiar feeling and that he could not describe it properly. It was especially marked in the left middle and ring fingers. He said: "When I do anything with that hand I feel as if I ought to be using the right hand."

*Examination July 9, 1940.*—Intelligent and co-operative. No aphasia. No disturbance of speech or writing. No apraxia. No disorder of recognition of objects.

Fundi normal. Left homonymous hemianopia. Slight weakness of left lower face. Spastic weakness of left upper and lower limbs, more pronounced in the lower than the upper, with increased tendon reflexes and diminished abdominal reflexes on the left side. Plantar reflexes flexor. Sensibility: Relative anæsthesia to light touch on the left half of the body, but touches when felt were accurately localized. Relative left hemi-analgesia, gross postural loss in the left fingers and toes, inability to recognize an object placed in the left hand and no threshold

for tactile discrimination on left hand. He was able to recognize his own and the observer's right and left hands and identify fingers named on his own and the observer's hands. No abnormal tension of bone flap.

*Subsequent history.*—Further X-ray treatment was carried out by Dr. Fairchild which led to considerable temporary improvement. In December, 1940, however, he was worse than he had been for six months previously. Dr. Fairchild reported that his cerebration was very slow, that he was disorientated both in time and space and quite helpless. A further course of X-ray treatment was begun, but he died shortly afterwards.

*Case 5.*—K. W. N., right-handed female, aged 47. Hypertension: cerebral hæmorrhage into posterior half of right cerebral hemisphere verified at autopsy. Difficulty in finding way about. At times tactile agnosia in both hands. Left homonymous hemianopia. Slight left hemiparesis and slight impairment of postural sensibility in the left fingers and toes.

*History.*—On July 11, 1940, she was walking along the pavement in which a hydrant standard was standing in a hole. She did not see it and caught her foot in the hole, falling and striking her right lower abdomen on the top of the hydrant. She does not think she hit her head nor was she unconscious. She was helped home by a workman and Dr. M. O'Regan saw her within an hour or two. She was then complaining of severe headache and photophobia and remained in a state of semi-consciousness and mental confusion with headache and vomiting for two or three days.

She was then sent into the New End Hospital, Hampstead, where she stayed for a week and was then discharged as she wanted to go home. On returning home she was still mentally confused and had visual hallucinations saying that she saw some dolls on the mantelpiece and that a little Chinaman came and sat on the end of her bed.

*Visual disorientation.*—She did not always recognize her room and it was noticed that she could not find her way about the flat. When she set out for the bathroom she arrived at the lavatory, which was a door on the right, and when she tried to go to the lavatory she made a similar mistake, took a turning to the right and got lost again. Yet when she was asked how she would find her way to the bathroom, the door of which was on the left at right angles outside her bedroom door, she replied: "I should go first to the cupboard in which my husband keeps his clothes." (This was near the bedroom door.) "Then I should open the bedroom door and outside would be where the coats are hung up. I should then look for the electric light switch which is outside the bathroom, because the Borough Council won't allow it inside, and I should then find the bathroom and the bath would be in it." Thus she clearly visualized the landmarks of the correct route. When asked to describe how she would find her way from the tube station to her flat she described this in detail correctly and apparently visualizing the landmarks, but she consistently said right instead of left for the turnings except on one occasion. When offered objects she tended to pass point to the right with both hands.

*Examination, August 1, 1940.*—Co-operative; slightly retarded mentally. Speech: She understood all that was said to her and was able to speak and read correctly except for occasional verbal mistakes. No agraphia.

Fundi normal. Left homonymous hemianopia. Pupils and ocular movements normal. Slight weakness of left side of face, left upper and lower limbs which were markedly hypotonic. Slight motor apraxia in left upper limb. No inco-ordination. Arm-jerks sluggish and equal; abdominal reflexes brisker on the right than the left side. Knee-jerks absent; ankle-jerks left brisker than right. Plantars flexor. Sensibility: No cutaneous anæsthesia; ? slight analgesia to pin-prick over left upper

limb. Appreciation of passive movement of the fingers and toes slightly impaired on the left side.

*Tactile agnosia.*—She was unable to recognize objects put into either hand, even when handling them with both as she always wished to do. Half-crown: "It feels like celluloid." A gold pencil: "This feels like celluloid too." A yale key: with the left hand nothing; with the right hand, fingering the hole, "A thing for knitting needles to test their size." An ordinary latch key, nothing. Large surgical scissors: "This is metal." She felt them with both hands and even when they were placed on the finger and thumb of the right hand and she made cutting movements she did not recognize them, but when she saw them said immediately: "A pair of blunt scissors." Medicine glass: handling it: "Something with a lip." She was then allowed to hold it to her lips and she made the movements of taking fluids. Afterwards she said "a milk jug," but she may have seen it. Recognized a hair brush, when the bristles were applied, with both hands and feet, but with some hesitation with the left hand.

*Distinction of right and left.*—She was able to name her own and the observer's right and left hands, fingers and feet with only occasional mistakes. She tended on August 1, however, to move the right arm when asked to move the left. When asked to touch her own nose and the observer's finger alternately she several times became confused between her own and the observer's nose.

Blood-pressure 210/150. Fading bruises around right anterior superior iliac spine. Cerebrospinal fluid pressure 215 mm., protein 80 mgm. Slight excess of lymphocytes.

*Subsequent history.*—Except for fluctuations her condition remained substantially the same for a month after the onset. During the latter half of August she gradually lapsed into unconsciousness and died on September 18 of bronchopneumonia.

Post-mortem examination by Dr. J. Davidson showed that the cerebral vessels were atheromatous. Section of the cerebral hemispheres showed an old cerebral hæmorrhage in the right hemisphere. It extended from the vicinity of the occipital pole to halfway between that and the frontal pole. The area was occupied with pale red blood-clot. The hæmorrhage had apparently arisen from the rupture of an atheromatous vessel.

*Case 6.*—E. Y., left-handed male, aged 53. Abscess of right parieto-occipital region secondary to bronchiectasis, verified at operation. Convulsion followed by visual disorientation. Difficulty in finding way about, turning to right instead of to left. Agraphia, alexia and ideational apraxia. Left homonymous visual field defect, later left homonymous hemianopia, left hemiparesis and loss of postural sensibility on left side.

*History.*—In July, 1940, he complained of cough, sweats, loss of weight and hæmoptysis since an attack of influenza the previous winter. X-ray examination of the chest showed a shadow in the right middle lobe near the hilum. Bronchoscopy and radiography after lipiodol injection showed bronchiectasis.

On March 2, 1941, he had a convulsion. He was standing just outside his house and "feeling funny" went indoors. He then became confused, felt "in the middle of a sort of maze," did not know where he was nor what to do and lost consciousness and fell. He became cyanosed, twitched on both sides of the body and foamed at the mouth. He was unconscious for fifteen minutes and semiconscious for half to three-quarters of an hour. He fractured his right clavicle in his fall.

After recovering consciousness speech was slow and hesitant. In reading he would miss out words and showed a tendency to read words from right to left, trying to pronounce "of" "fo" for example. When given coffee on his right hand

and bread on his left he took up the bread and tried to drink it. He tried to eat an egg like an apple, putting the shell in his mouth. He was unable to fill his pipe. During the next week he suffered from headaches of increasing severity, vomited once and was sometimes drowsy.

*Visual disorientation.*—To go from his bedroom into his sitting-room involved crossing the hall and entering the left hand of two doors facing him. The door to the right led into the drawing-room. In trying to do this he went into the drawing-room, but at once recognized his mistake and became irritated by it. On returning to his bedroom in which there were two beds placed head to foot facing him he got into the right hand one instead of the left which was his own bed. His wife had noticed his tendency to go to the right in walking.

*Examination, March 12, 1941.*—He was left-handed as a child, and still used his left hand for cutting bread and dealing cards, but wrote with his right hand. Alert and co-operative, and orientated in place and time. In reading, words were omitted and there was paraphasia. He said that he felt an impulse to read from right to left. He wrote his name correctly, but when asked to write "God Save the King" there was paragraphia. He understood spoken speech and named objects correctly. He used common objects correctly, but when asked to carry out more complex actions there was severe ideational apraxia. The optic discs were normal. There was visual inattention in the left half-fields. Though the fields appeared full on confrontation, perception of movement was defective in the left half-fields, and two days later there was a left homonymous hemianopia. There was no disorientation in pointing with the eyes closed to objects on either side which he had previously been allowed to see, nor in grasping objects. He identified his own and the observer's right and left hands correctly after a little hesitation. At first the only other abnormality was a relative exaggeration of the tendon reflexes and diminution of the abdominal reflexes on the left side, but two days later there was a hemiparesis with impairment of postural sensibility on the left side and astereognosis in the left hand.

On March 15 Mr. Northfield operated and found an abscess fairly near the surface through a burr-hole just below and behind the right parietal eminence. There was no capsule and the brain was extremely soft. He removed 10 c.c. of thick pus. A second aspiration was made three days later and the patient made an uninterrupted recovery. When examined on May 30 he complained of no symptoms and showed no abnormal physical signs. On July 28, 1941, he described by letter two brief attacks of disorientation probably epileptic equivalents.

These patients, Nos. 4, 5 and 6, thus exhibited quite a different form of disorientation from those in the first group. As Nos. 4 and 5 had a complete homonymous hemianopia when examined, their power of visual localization in the affected half-fields could not be tested. No. 6 ultimately developed a complete left homonymous hemianopia, but when first seen he was able to see objects in his left half-field and then made no mistake in pointing to them or grasping them such as patients Nos. 1, 2 and 3 made. No. 5 tended to pass point to the right with either hand when trying to grasp objects presented to her seeing half-fields. This was probably due to the displacement of the fixation point to the seeing side in hemianopia described by Fuchs. They showed no loss of topographical memory, no gross difficulty in recognizing their own or the observer's

right and left sides, and no visual agnosia for objects. One patient, No. 4, had the greatest difficulty in dressing himself; the other two always had this done for them. All three showed some defect of postural sensibility on the left side.

A few similar cases are to be found in the literature, though the nature of the disorder of route-finding does not seem to have been fully recognized. Hughlings Jackson (1876) reported a case of tumour of the right temporo-occipital region causing left hemiplegia and "imperception." The patient, though her topographical memory was well preserved, got lost in familiar surroundings and made mistakes in dressing herself. Wendenburg (1909) described a case of tumour of the right occipital lobe in a man whose first symptom was inability to find his way about, and who also had great difficulty in dressing himself. Finally, he became completely disorientated in space. In two cases (Wilbrand, 1892, and Pötzl, 1928) the syndrome was complicated by concomitant lesions of the left hemisphere, but in Pötzl's case the left hemisphere lesion was relatively unimportant.

It is clear that the disorientation cannot be attributed either to visual inattention or to hemianopia in the left half-fields. Patients with hemianopia may run into objects, but they do not get lost in their own homes. Evidently we have to do with a disturbance of spatial orientation and not with a mere inability to see normally. It is also clear that the disorder is not due to a loss of topographical memory, nor to a visual agnosia which made it impossible to recognize the surroundings, for patients who arrived at the wrong door recognized that they had made a mistake.

Pötzl develops an elaborate theory of the organization of space perception which may be true, but for which the single reported case seems rather a slender basis, especially as several different forms of visual disorientation are encountered. Nevertheless, his interpretation of his patient's disorder of route-finding appears correct in essence, though the three cases with lesions confined to the right hemisphere reported in this paper permit a more precise statement of it. Pötzl explains this symptom as a disorder of orientation in surrounding space arising from an error of discrimination of right and left turning. He considers that an automatic route, with all its necessary right and left turns, is centrally represented as a whole, somewhat like a sentence in the realm of speech or an object or a total situation in the concrete perceptual external world. This unitary route becomes damaged in its coherence and liable to become confused with others like the words in aphasia or the representations of objects in object-agnosia. This may be true of some disorders of route-finding, but the explanation in the cases now under

consideration is simpler. The failure in the choice of right and left turning is not indiscriminate. The effect of a lesion of the posterior part of the right hemisphere is to cause the patient to neglect the left half of external space and to select a right instead of a left turning, or even the right hand of two doors or other objects facing him, in error. This mistake extended even to No. 5's description of her route from the Underground Station to her home. The patient's behaviour towards the left half of external space is similar to the attitude adopted towards the left half of the body by some patients in whom awareness of the body is disordered. Since each half of the body is a part of the corresponding half of external space, it is not surprising to find that perception of the body and perception of external space are closely related and subject to similar disorders.

### Visual Disorientation and the Body Scheme

Three patients out of the six in this series experienced some positive disorder of awareness of the half of the body corresponding to the side on which visual orientation was disturbed, and all showed defective postural sensibility on that side. Anatomically this is explained by the situation of the lesions in the parietal lobe between the visual cortex and the postcentral convolution; physiologically by the rôle of this region in both spatial orientation and awareness of the body. The postcentral convolution is concerned with crude sensations of movement and posture derived especially from the joints: the visual cortex with the perception of objects external to the body. The surface of the body is a frontier which is perceived from both sides, by vision as part of the external world and from within by the various forms of cutaneous sensibility. The visual localization of an object in the external world implies in the first instance the power to relate its position to that of the body also perceived as a visual object. To ask which is the nearer of two objects has no meaning except in relation to the position of the observer's body. Visual localization is thus a process involving the body scheme and the scheme of the external world. This interrelatedness of spatial orientation and awareness of the body implies an anatomical relationship, which is found in the parietal lobe, between the visual cortex and the postcentral convolution (Pötzl, 1928; Nielsen, 1938*a*). Both these functions, therefore, are likely to be disordered together. Two out of the three patients who had difficulty in localizing objects visually in homonymous half-fields complained that the ipsilateral half of the body felt as if it did not belong to them. Those patients, Nos. 4, 5 and 6, in whom awareness of the left half of the external space was more profoundly disordered showed a spatial disturbance of the same

kind as that observed in some patients with disordered perception of the left half of the body. Nielsen (1938*a*), discussing the physiological mechanism of the body scheme in the light of his own previously published cases, maintains that the essential lesion is one involving the right thalamo-parietal bundle which prevents sensation from reaching the supramarginal cortex. The patient who is thus cut off from the sensations which are necessary for the construction of a body scheme may react to the situation in several different ways. He may remember that the limbs on his left side are still there, or he may periodically forget them until reminded of their presence. He may have an illusion of their absence, i.e. they may "feel absent" although he knows that they are there; he may believe that they are absent but allow himself to be convinced by evidence to the contrary; or, finally, his belief in their absence may be unamenable to reason and evidence to the contrary and so constitute a delusion. In anosognosia the patient is unaware not that a part of the body exists, but that it is diseased: he is unaware of hemiplegia, blindness or deafness.

In my patients Nos. 4, 5 and 6 the patients' attitude to the left half of external space was one of inattention or neglect: in walking he behaved as if it were not there, but in conversation he recognized its existence. His state was thus comparable with an amnesia for the left half of the body. Both were present in Pineas' (1931) patient, who failed to perceive the left half of his own body and also, though there was no hemianopia, the left half of objects, persons and pictures in external space. Allæsthesia for sounds was associated with anosognosia in a patient of Dr. Parkes Weber's whom he kindly allowed me to see and whom he is reporting more fully elsewhere.

A man, aged 53, as a result of multiple cerebral emboli, was completely blind and had a left hemiplegia with gross loss of all forms of sensibility on the left side of the body. Sensory stimuli applied to the left half of the body were sometimes referred to the right. He always denied both his blindness and his hemiplegia. When he grasped his left thigh with his right hand he thought it was the urinal. His head and eyes were usually turned to the right. His hearing was acute in both ears, but he referred sounds heard with his left ear to the right side pointing to the right with his arm fully abducted. He was agnosic for everyday sounds, but not for speech, and understood what was said to him. As he was bedridden, his powers of orientation could not be further tested. The association of agnosia for the left half of the body, anosognosia for left hemiplegia and allæsthesia for sensory stimuli and for sounds heard with the left ear indicate a severe disturbance of awareness of the left half, both of the body and of external

space. This case is very similar to Nielsen and Sult's (1939) patient No. 5 who, amongst other symptoms, had anosognosia for total blindness and left hemiplegia, and acoustic agnosia for sounds but not for spoken speech.

But my patients not only failed to turn to the left; they chose the right in error. This mistake is physiologically comparable with allæsthesia in patients in whom the body scheme on one side of the body is disordered. In such patients not only may a stimulus applied to the affected half of the body be felt upon the normal side, but voluntary movements spontaneously initiated may feel as if carried out on the normal side as in Nos. 1, 2 and 4, or the whole affected half of the body may feel transposed, as in No. 4. It appears that severe damage to the scheme for one half of the body causes events occurring on that half, if perceived at all, to be related in consciousness to the surviving scheme representing the normal half. Similarly, severe damage to the scheme for one half of external space causes the remaining half to exert a dominant influence in the choice of routes.

Furthermore, a severe disturbance of awareness of the left half of space may so disrupt spatial perception that the patient fails to recognize his surroundings. This occurred at some stage in the illness in Hughlings Jackson's case, Wendenburg's case and my Cases 3, 4 and 5, in all of whom the lesion was confined to the right cerebral hemisphere.

### Apraxia for Dressing.

Two patients in this series exhibited apraxia for dressing, a symptom which does not appear to have been specifically described hitherto. No. 1, whose lesion was in the upper and mesial part of the left parietal lobe, complained that she had great difficulty in putting on her clothes, and might put them on back to front or upside down. She attributed this to the fact that she did not know the right side of a garment from the left. No. 4, whose lesion was in the right temporo-parietal region, said that it was impossible for him to put his clothes on in an automatic fashion. In order to put on his pyjama coat he had to lay it out on the bed and then utilize his knowledge that the pocket was on the left side and the buttons on the right. Hughlings Jackson's patient, with a right temporo-parietal tumour, made mistakes in dressing herself; "put her things on wrong side before." Wendenburg's patient, with a tumour in the same situation, put his clothes on upside down and his legs into his coat sleeves. He did not know which was the inside and which the outside of his coat, and when given a shirt turned it over in his hand and was quite unable to put it on. Neither of my patients showed apraxia in any other activity,

nor object-agnosia. No. 1 could not name her own or the observer's right and left sides, but No. 4 could do so. They had in common a defective awareness of one half of external space and of the same half of the body, with spontaneous allæsthesia, in the one case on the right and in the other on the left side, and both were right-handed.

These facts show that apraxia for dressing, though no doubt it may occur as one manifestation of a more general ideational or motor apraxia, may be an isolated form of apraxia. In putting on a garment we dispose it in space in the way that will best facilitate the introduction of an arm or a leg. In this process, from long habit, the visual images automatically evoke the appropriate movements, and the right or left side of the garment is brought into relation with the right or left side of the body without any conscious consideration of right and left or the use of these symbols. When the patients said that they were unable to recognize the right and left sides of a garment they meant that seeing the garment no longer evoked a knowledge of its spatial relationship to the body, and so did not lead to the appropriate actions. This disorder is an ideational apraxia. It is independent of the inability to name the right and left sides of the body, for this, though present in No. 1, was absent in No. 4. No. 4, however, was able to use these verbal symbols to escape from his difficulty: by identifying and naming the right and left sides of his pyjama coat he was able to put it on correctly, though he could not do so "automatically." Apraxia for dressing is also independent of constructional apraxia, though clearly a very similar disorder. It appears closely related to the disorder of the body scheme for one half of the body, for the recognition of the structure of clothes must necessarily be intimately related to the perception of the body. Hence a lesion in either hemisphere which disorganizes the body scheme may cause apraxia for dressing.

### Visual Disorientation and Cerebral Dominance.

Recent work has shown that the problem of the dominance of the left cerebral hemisphere is much more complex than had been thought. The discovery that symbolic thinking and expression are normally functions peculiar to the left hemisphere led to the view that these functions had been superimposed on the pre-existing functions of this hemisphere which otherwise might be expected to be still the mirror image of the right. However, it has been found not only that the right hemisphere may sometimes be dominant for some functions normally exercised by the left in right-handed persons, but also that certain syndromes, notably anosognosia, are seen exclusively or almost exclusively as a result of lesions

in the right hemisphere. To this last group, although the number of reported cases is yet small, it may be necessary to add amnesia or agnosia for one-half of external space, since in all the reported cases in which this disturbance can be recognized the lesion has been in the right hemisphere.

The cases reported in this paper show that visual localization in external space is not a function to which dominance applies. Riddoch (1935) showed that defective visual localization limited to the right half-fields could follow a lesion of the posterior part of the left hemisphere. My Cases 1 to 3 confirm this and show that the same is true, reciprocally, of the left half-fields and the right cerebral hemisphere in right-handed persons.

Nielsen (1938*b*) compares the disturbance of the body scheme resulting from lesions of the two hemispheres and points out that lesions of the major, dominant, hemisphere cause disturbance of knowledge of the two hands (Gerstmann's syndrome), while lesions of the minor hemisphere lead to "a dropping out of the opposite side of the body, first from attention and then from consciousness." He discusses Herrmann and Pötzl's (1926) ambidexterous patient in whom a lesion of the right hemisphere caused both Gerstmann's syndrome and amnesia of the left half of the body, the transfer of dominance to the right hemisphere having caused it to behave as both the major and the minor side. My patient No. 6 is an example of dissociation of functions in relation to dominance. He was left-handed and his development of alexia, agraphia and ideational apraxia showed that for purposes of symbolization the right was the dominant hemisphere. Nevertheless, he showed no visual object-agnosia and no loss of topographical memory, but exhibited the form of spatial disorientation characteristic of a lesion of the minor hemisphere. Thus there was no evidence that the shift of dominance for symbolic functions to the right hemisphere converted that hemisphere into the major one for spatial orientation. Nielsen (1937, 1940) believes that a similar dissociation may occur in respect of visual recognition of objects and that visual agnosia may be produced by a lesion of the right occipital lobe in a right-handed person whose left hemisphere is the dominant one for speech functions. It would seem, therefore, that symbolic thought and expression, visual recognition of objects, visual orientation in space, topographical memory (Nielsen, 1940) and awareness of the body are functions which behave independently of one another in relation to hemisphere dominance, and that in the case of any one of them the functions normally performed by the left hemisphere may be transferred to the right without the occurrence of a similar shift in the others.

## The Nature and Classification of Visual Disorientation.

The term visual disorientation may now be seen to be a loose and comprehensive description covering a number of disorders of function differing in their nature. In the light of the cases reported in the literature and in this paper an attempt will be made to distinguish and classify these.

(1) Defective visual localization of objects in space. This exists in three main forms: (i) As a result of impaired visual acuity due to lesions of the afferent visual pathways; (ii) in homonymous hemianopia, owing to displacement of the fixation point; and (iii) as a result of damage to that integrative process whereby visual perception evokes awareness of the spatial relations of objects seen. The last disorder alone of the three is a true visual agnosia; it is an agnosia for spatial relationships among objects seen. Kleist calls it space-blindness and Pötzl includes it among his geometrical object-agnosias. It is characterized by an inability to appreciate the absolute and relative position of objects seen, especially in relation to the patient's sagittal plane. This may cause disordered perception of relative size and movement. This agnosia for spatial relationships is independent of defects of visual acuity. When it is the result of a lesion of one cerebral hemisphere it is limited to the opposite homonymous half-fields and, its features being the same whether it occurs on the right or the left side, it is unrelated to hemisphere dominance. Bilateral lesions cause much more severe disturbance than unilateral lesions, because the patient with a unilateral lesion, though inconvenienced, can still orientate himself through his remaining half-fields. He may run into objects because he misjudges their distance, but he can still find his way from one place to another. Bilateral lesions may completely destroy awareness of the absolute and relative positions of objects and hence orientation among them. Such patients are space-blind, whereas those with a unilateral lesion are only space-hemianopic. Patients who are thus space-blind cannot find their way round objects when they run into them, set out in the wrong direction to get to objects which they clearly see, and have difficulty in finding their way about and learning the topography of a room.

Defective visual localization of objects in space appears to be the result of a lesion of the upper part of the parietal lobe towards the vertex, and there is slight evidence that a lesion in the temporo-parietal region may selectively affect orientation in space within reach of the hand. Defective visual localization confined to the vertical plane is extremely rare. I have seen one example which may be mentioned, though since the lesion was

unverified it throws no light on the physiological pathology. To this patient objects above her head appeared lower than they actually were; for example, in hanging clothes on the line she missed the line by some inches on the near side. She made errors above and below when pointing to objects above the horizontal plane.

(2) Loss of stereoscopic vision. Loss of stereoscopic vision as in Riddoch's (1917) and Holmes and Horrax's (1919) case is extremely rare. Holmes and Horrax point out that it cannot be due to defective recognition of distance nor to defective accommodation, since either may be present without it; nor is it the result of loss of appreciation of light and shade, which was not defective in their patient. They attribute it to a failure to fuse the non-corresponding images formed in the two eyes by a solid object. However, this cannot be the sole factor, since monocular vision is not entirely astereoscopic. The significant fact is that in cases of visual disorientation the thickness of objects can usually be appreciated even though the distance between them in a sagittal plane—thickness of space—cannot. Evidently, therefore, our awareness of the spatial qualities of objects is a function anatomically independent of our awareness of the spatial framework in which they lie. Loss of stereoscopic vision is disordered awareness of visual-object-space. In Holmes and Horrax's case the lesion was believed to involve both angular gyri.

(3) Agnosia for the left half of space. Patients with a massive lesion of the right parieto-occipital region exhibit a tendency to neglect the left half of external space and in consequence when following a familiar route to turn to the right instead of to the left in error. No corresponding disturbance has been reported as a result of a lesion of the left hemisphere. This is a more severe disorder than the agnosia for spatial relationships among visual objects: it is an agnosia for external space as such. It resembles the impairment of perception of the left half of the body which is produced by lesions of the right parietal lobe.

(4) Visual allæsthesia. This rare disorder is mentioned for the sake of completeness. It consists of a transposition of visual images from one half-field to the opposite one, and sometimes from the lower to the upper quadrant at the same time. It was first observed in migraine, but has also been reported as a symptom of a lesion at one or both occipital poles. Herrmann and Pötzl (1928) interpret it as the outcome of two factors, a partial blocking of centripetal visual pathways and the loss of a specific direction factor. It is thus comparable with tactile allæsthesia and is a manifestation of a similar process to that which causes the erroneous right turning described in the previous section.

(5) Loss of topographical memory. In spite of any of the disabilities already described, a patient may be able accurately to describe routes familiar to him and to visualize familiar scenes. Loss of this ability, topographical memory, is a disorder independent both of other forms of visual disorientation and of loss of memory for objects. Loss of topographical memory was present in cases reported by Wilbrand (1892), Pötzl (1928), Holmes (1918), Holmes and Horrax (1919), in all of which lesions were bilateral. However, the available evidence suggests that loss of topographical memory is usually the result of a left occipital lesion, the left being the dominant hemisphere for this function, though the right may be so occasionally (Nielsen, 1940).

(6) Visual disorientation secondary to visual object-agnosia. It has long been known that patients with visual object-agnosia have difficulty in finding their way about. von Stauffenberg, in his review of mind-blindness, points out that visual disorientation is constant. He attributes it to a defect of visual impressions which possess too little distinctness to awaken the visuo-motor engram series necessary for orientation. Such patients often recover their power of orientation in familiar surroundings, which von Stauffenberg explains by saying that movements acquire the power to evoke the mnemic quasi-reflex dispositions upon which orientation depends. Confronted with new tasks, however, the visual conception of the mind-blind person is too feeble to permit the registration of spatial signs. All these features were well shown by a recent patient of my own (Brain, 1941).

(7) Mixed forms. Only by the investigation of patients with localized unilateral lesions has it been possible to analyse the various forms of visual disorientation. Patients with multiple lesions, whether arteriosclerotic or traumatic, exhibit all possible combinations of the symptoms described above in a confusing welter of disorder, as in the cases observed during the last war. A satisfactory analysis of these should now be possible.

(8) Unspecific symptoms. It will be apparent that certain symptoms may arise from more than one form of visual disorientation. Two of these which may be specially mentioned are disorder of route finding and disorientation as to place. The following may render a patient unable to find his way about: (i) Inability to localize objects in both halves of space; (ii) Agnosia for the left half of space. (iii) Loss of topographical memory. (iv) Visual object-agnosia. All of these disorders are produced by lesions in the parietal or occipital lobes. (v) Lesions elsewhere in the brain, especially perhaps in the frontal lobe, may impair the patient's ability to utilize the parieto-occipital functions even though these may themselves

be undamaged (Raney and Nielsen, 1940), as in some cases of dementia. Similarly, to say that a patient is disorientated as to place is to say little: it is necessary to discover what impairment of function is the cause.

(9) Secondary symptoms. There are numerous symptoms which are associated with visual disorientation and which, though of considerable interest, can only be mentioned here. These include disorder of the ocular movements and accommodation, loss of visual attention and of the menace reflex, and difficulty in judging the relative sizes of objects, counting things by vision, and reading and writing (Holmes, 1918).

Finally, it is necessary to add that visual disorientation involves more than the mere inability to orientate oneself by means of vision. Though we perceive space by a number of different senses, vision is so much the richest in spatial data that visual imagery is often a component of the spatial percepts evoked through other sensory channels. Hence severe damage to the central processes of space-perception on the visual side deranges the whole basis of orientation, and, as several writers have pointed out, patients with visual disorientation are far more severely handicapped than a blind man, and lack a blind man's capacity to orientate himself by other senses. My patient No. 4 was even more disorientated in the dark than in the light. Hence just as space, through whatever sense we perceive it, is one, so our awareness of it is not a mere juxtaposition of sensory impressions from which some can be deducted without impairing the validity of the remainder: it is a synthesis from which springs a unity of perception corresponding to the external reality.

## Summary.

(1) Three cases are reported of visual disorientation limited to homonymous half-fields, in one case on the right and in two on the left side.

(2) This form of visual disorientation which results from a lesion of the opposite parietal lobe is not due to a defect of visual acuity, but is regarded as a defect in visual localization of objects in the affected half-fields.

(3) Three cases are reported of massive lesion of the right parietal lobe causing inability to follow familiar routes, owing to a selection of right instead of left turnings in error.

(4) This disorder is attributed to an inattention to the left half of external space, comparable with the amnesia for the left half of the body, which may also follow a lesion of the right parietal lobe.

(5) Two cases of apraxia for dressing are described and the relation of this symptom to disorder of the body scheme is discussed.

(6) Visual disorientation is considered in relation to the dominance of one cerebral hemisphere.

(7) The nature and the classification of the various forms of visual disorientation are discussed.

I am much indebted to Mr. D. W. C. Northfield for kindly referring Case 1 to me and for his surgical collaboration in several of the other cases, to Major P. B. Ascroft for making available to me his notes of Case 4 and to Dr. Parkes Weber for allowing me to see and allude to his patient.

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# AKINETIC MUTISM WITH AN EPIDERMOID CYST OF THE 3RD VENTRICLE.

## (WITH A REPORT ON THE ASSOCIATED DISTURBANCE OF BRAIN POTENTIALS.)

BY H. CAIRNS, R. C. OLDFIELD,[1] J. B. PENNYBACKER AND D. WHITTERIDGE.[2]

*(From the Nuffield Department of Surgery, Oxford.)*

IN a case of cystic tumour of the 3rd ventricle we have observed a peculiar mental state which may be described as akinetic or trance-like mutism. An incomplete variety of this condition has also been observed after ventriculography in chronic hydrocephalus due to stricture of the aqueduct of Sylvius and other lesions of the mid-brain. In this paper, to assist recognition of the clinical state and also to bring forward clinical evidence relating to the influence of the diencephalon on the activity of the cortex and on consciousness, we report in detail the case of 3rd ventricle cyst.

The clinical picture of the condition is as follows. The patient sleeps more than normally, but he is easily roused. In the fully developed state he makes no sound and lies inert, except that his eyes regard the observer steadily, or follow the movement of objects, and they may be diverted by sound. Despite his steady gaze, which seems to give promise of speech, the patient is quite mute, or he answers only in whispered monosyllables. Oft-repeated commands may be carried out in a feeble, slow, and incomplete manner, but usually there are no movements of a voluntary character; no restless movements, struggling, or evidence of negativism. Emotional movement also is almost in abeyance. A painful stimulus produces reflex withdrawal of the limb, and, if the stimulus is sustained, slow feeble voluntary movement of the limbs may occur in an attempt to remove the source of stimulation, but usually without tears, noise, or other manifestation of pain or displeasure. The patient swallows readily, but has to be fed. Food seen may be recognized as such, but there is evidently little appreciation of its taste and other characteristics: objects

[1] From the Psychological Laboratory, Cambridge, and working with a Rockefeller Research Fellowship.

[2] Working with a Beit Research Fellowship.

normally chewed or sucked may be swallowed whole. There is total incontinence of urine and fæces.

Fluctuations may occur in the intensity of this state. In its incomplete manifestations the patient may respond at times, though slowly and imperfectly, by speech and voluntary movement. Voluntary movement may be accompanied by a coarse tremor of the limbs. Incontinence persists, and there is little or no trace of spontaneous activity or speech. The onset of the condition is gradual. In certain circumstances there may be slow spontaneous recovery; but if the state is caused by pressure of a tumour it may be followed by coma. The condition is sometimes, though not always, associated with decorticate rigidity.

### Report of Case.

*Epidermoid (Rathke Pouch) Cyst of 3rd Ventricle. Akinetic Mutism, relieved by Aspiration of Cyst.*

*History.*—Elsie Nicks (No. 7081), a girl aged 14, was admitted to the Radcliffe Infirmary on March 2, 1940, complaining of attacks of headache. These had begun eight years before, and each was preceded for a few days by listlessness. The pain was over the left eye and was associated with a feeling of pins and needles in the right side of the face, the appearance of coloured lights and stars in the left half of the visual fields, and often with vomiting. At times central vision also was obscured. The attacks lasted about two days, and recently had become so intense as to cause her to scream with the pain. Since infancy she had suffered from occasional enuresis, and she had grown very little in height since the age of 9. She was right-handed.

*Examination.*—She was a plump, short child (height 4 ft. 3½ in., weight 5 st. 10 lb.) with thin eyebrows and a pale, finely wrinkled and delicate skin. Temperature 97°, pulse-rate 80, respiration rate 20. Her bearing and behaviour appeared to be that of a child of 9, but her mental age on the Revised Stanford-Binet intelligence test, Form L, was 12 years and 3 months. Her head was enlarged (54 cm. circumference). Both optic discs showed primary optic atrophy. The visual acuity was $\frac{6}{9}$ in each eye and her visual fields were full. The central nervous system was otherwise normal. There was complete absence of secondary sexual characteristics. In the long bones the condition of the epiphyses corresponded to that of a child between 9 and 12 years of age. Her sugar tolerance was raised; her basal metabolic rate and fluid exchange were normal. X-rays of the skull showed a calcified mass within the sella turcica (fig. 1) and the usual signs of raised intracranial pressure.

The diagnosis was that of tumour of Rathke's pouch. Her headaches subsided with rest, and as there was no evidence of involvement of the optic chiasm it was decided to defer operation.

*Subsequent course.*—Within a few weeks headaches returned. They became so intense that she was given morphia. At the height of each attack she would vomit several times and complain of defective sight. Her mother said that she had lost the strength of her limbs, that her speech had become affected, and that she was very drowsy. She was readmitted on April 25, 1940, in a state of apathy and intermittent mutism. She would occasionally whisper a short but relevant answer to a question, or move her limbs to command; but it was impossible to keep her attention

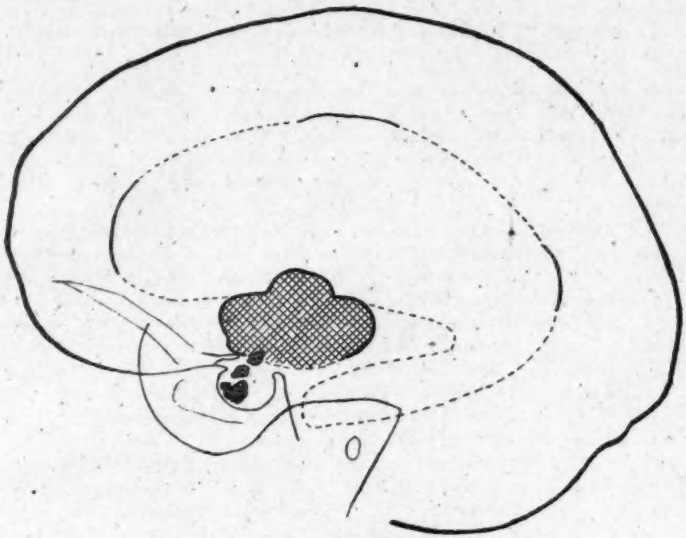

FIG. 1*a*.

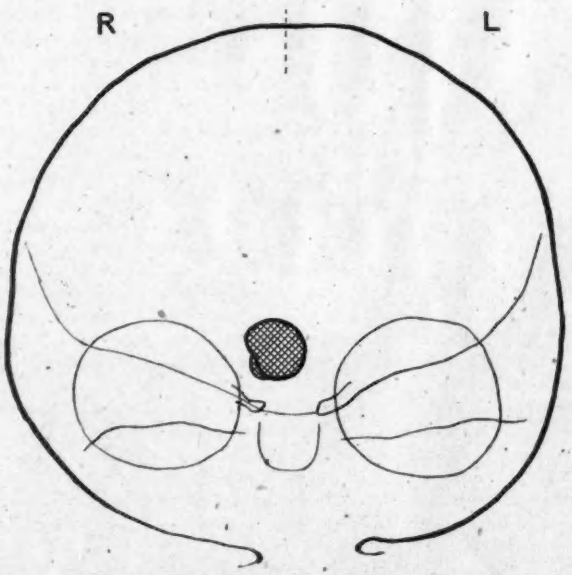


FIG. 1*b*.

FIG. 1 (*a*) and (*b*).—Suprasellar cyst (cross hatched) and lateral ventricle outlined by a series of X-ray pictures of an air bubble injected after aspiration of the cyst. The continuous line shows the actual outline, the dotted lines are an estimate of the outline not actually seen. In the lateral view patches of intrasellar calcification are also represented.

thus employed for more than a few seconds at a time. She lapsed not into sleep but into a state of inertia and silence. All four limbs were moved weakly but without tremor. The abdominal reflexes were no longer present and the plantar reflexes were now extensor in type. Sensibility appeared to be preserved, but with high threshold. Her peripheral circulation was poor.

In the following days she became less accessible. She did not sleep excessively. When awake and undisturbed she made no movement, save that her eyes would follow quite alertly any movement in the ward, and on a few occasions she whispered, almost inaudibly, a monosyllabic response. For the most part she made no reply to urgent appeals, though on one occasion she gave a feeble smile when tickled, and turned her head away. She had to be fed. When a chocolate had been placed in her hand she attempted to put it in her mouth, but she dropped it and did not try to reclaim it. However, she then made some tentative chewing movements. When barley sugar was placed in her mouth it was immediately swallowed whole, and without any display of interest or discomfort. Sugar and quinine were swallowed with complete indifference. Sustained pin-prick on the trunk evoked a slow, deliberate movement of both hands towards the stimulus, but no sound, grimace or tears, though once or twice her expression changed a little as though she were on the point of crying. There was no trace of catatonia. She was totally incontinent of urine and fæces.

*Ventricular tapping.*—On April 28, three days after admission, she had four attacks of extensor rigidity at short intervals, with limbs extended, head retracted, pulse and respiration slowed. Between the attacks extensor tonus remained excessive and she could not be roused. The lateral ventricles were tapped through the left frontal and right parietal regions, and were found to be dilated, with an initial pressure of over 600 mm. $H_2O$. After release of the ventricular pressure she could be roused and her general condition improved, but the state of akinetic mutism was little affected. In the next few days she had slight and temporary right hemiparesis. When she was awake, her eyes still followed every movement of an observer approaching her bedside, and seemed always to give promise of speech. But repeated attempts usually failed to elicit any utterance. During sleep her eyes were normally closed. On May 6 she had a further attack of extensor rigidity with opisthotonos lasting two to three minutes. On May 9 she responded to strenuous appeals for a short period, recognizing one of us by name in a whisper, naming the hospital, and moving her limbs to command, albeit slowly and incompletely, but it was impossible to keep her attention for more than a few moments at a time. At this stage the right hemiparesis was less; in the face, it was seen to diminish as she wakened.

*First aspiration on 3rd ventricle cyst.*—On May 9 the right lateral ventricle was tapped through its frontal horn. The initial pressure was 190 mm. $H_2O$. The needle was advanced towards the anterior part of the 3rd ventricle, and after slight resistance a cyst was entered and 18 c.c. of brownish fluid were aspirated, containing 2·8 per cent. of protein and also cholesterol crystals. Five c.c. of air were injected into the cyst and subsequent X-rays showed that the cyst occupied the greater part of the 3rd ventricle (fig. 1).

Immediately the cyst was tapped the child roused and made a noise, the first loud sound she had made since admission to hospital two weeks before, but she would not speak. Within ten minutes, however, she gave her name, age, and address correctly without any trace of dysarthria, and then asked where she was. She co-operated perfectly in an examination. Within the next week the right hemiparesis disappeared, and she got up. She was still at times incontinent. She had

no memory of the two weeks in hospital before the cyst was emptied. She was discharged on May 22, at which time, apart from a defect of sustained attention, she was alert and intelligent and showed no neurological signs other than optic atrophy and occasional incontinence of urine.

In June, 1940, she began once more to have attacks of headache, vomiting, and impaired sight. In August her visual fields showed for the first time an incomplete upper quadrantic bitemporal hemianopia, but her visual acuity remained normal. She continued to be incontinent of urine at nights.

On October 17, after a severe attack of headache and vomiting lasting several days, she became once more mute and was readmitted to hospital in a dehydrated state. Her condition was similar to the state observed in April. She was more drowsy than on previous occasions, but could be awakened, when she would watch the movements of people in the ward. Addressed in a loud voice she would move her eyes towards the sound. There was a sluggish menace reflex. She swallowed without difficulty and would withdraw from painful stimuli. She seemed to understand at least something of what was said to her: for example, she would at times make feeble attempts to put out her tongue on command; but she was completely devoid of speech, and her only emotional expression was a feeble grimace in response to strong painful stimuli. She was incontinent of urine and fæces. Her pupils were irregularly small. Her extremities were cold and cyanosed. Her upper limbs always assumed a position of flexion, her lower limbs were extended and showed strong extensor tonus. The ankle-jerks were increased, there was bilateral ankle clonus, and both plantar reflexes were extensor.

She became stuporous and had to be fed by nasal tube. At 4.30 a.m. on October 19 she had an attack of extensor rigidity of the limbs and trunk with head retraction and upward deviation of the eyes. This lasted two minutes and was followed by coma. The pulse-rate slowed from 80 to 64. Respirations slowed from 20 to 10, and were deep and stertorous.

*Second aspiration of cyst.*—On October 19, seven hours after the attack, the cyst was aspirated again through the right frontal horn, and 15 c.c of brownish fluid were removed (initial ventricular pressure 80 mm. $H_2O$). Immediately after the aspiration, even before the needle was withdrawn, she became more alert and started moving about in bed. She then sat up, as though waking from deep sleep. She took a bite from a biscuit but would not take it into her own hand. Within half an hour, during electro-encephalography, she exclaimed, "Oh, dear" a number of times in a stereotyped fashion, but made no other verbal response. Two hours after the cyst had been emptied she began to talk, and an hour later she rang for a bed-pan. For two days she was slow in answering questions and mildly confused, but she helped herself to food with obvious enjoyment. She named objects slowly but without error, and could cut with scissors, though somewhat clumsily. Her amnesia extended from some days before to several hours after aspiration of the cyst.

She soon became bright and cheerful. With the improvement in mental state all signs of extensor rigidity disappeared. Her tendon-jerks, abdominal and plantar reflexes were normally present. She was discharged from hospital on October 23.

*Third aspiration of cyst.*—She was readmitted on November 17 after a particularly severe attack of headache, with which was associated incontinence. She was drowsy, but could be easily roused and would talk in response to questions, though she did not volunteer any remarks. Her abdominal reflexes were absent, but there were no other neurological signs in the limbs or trunk. On November 18 the cyst was tapped again, and 12 c.c. of brownish fluid were removed (initial ventricular pressure of cerebrospinal fluid, 220 mm. $H_2O$). Improvement was slow,

but within forty-eight hours she was once more moderately alert. Further surgical treatment was deferred on account of a left otitis media.

*Operation.*—On December 17, 1940, the 3rd ventricle was explored by resection of part of the right frontal lobe and a transventricular approach. A thin-walled yellowish cyst presented at the foramen of Monro. It occupied the 3rd ventricle and was in places adherent to the walls by vascular adhesions, especially at the lamina terminalis and the anterior part of the medial surface of each thalamus. In the floor of the 3rd ventricle there was a solid portion of tumour; this was not removed, but the cystic part was dissected away completely.

*Pathological report* (Dr. Dorothy Russell).—The specimens received for histological examination included two larger portions, measuring 3 by 1 by 0·3 cm. and 3·5 by 1 by 0·5 cm. respectively, and one smaller portion, measuring 0·7 by 0·5 by 0·4 cm., of soft yellowish grey tissue. The larger bits bore in places a smooth glistening surface to which some cholesterol crystals were attached. The remaining surfaces were firm and nodular.

In addition there was a portion of cerebral cortex and white matter measuring 4 by 2·5 up to 3·8 cm. deep. This was macroscopically normal apart from operative trauma, and was not examined microscopically.

*Microscopically.*—The tissue from the 3rd ventricle is composed, for the most part, of neuroglial cells which are mostly of pilocytic form. Hyaline degenerative changes are present in many of them. The surface bears, in many places, a well-differentiated epithelium of basal type composed of polygonal cells and, in relationship with the neuroglia, a regular layer of columnar cells. Papillæ composed of these cells penetrate the neuroglia for a considerable depth in some places. Superficially the epithelium frequently contains squamous and horny nests. The latter are occasionally present in the neuroglial tissue and may then be associated with a foreign body giant-cell reaction.

*Post-operative course.*—After operation there was a recurrence in mild degree of the mental state seen at earlier stages. In the first few days her speech was mainly limited to "Yes" or "No." She moved her limbs to hold a drinking cup, to rub her nose, or to withdraw from painful stimuli, but not in response to commands. Her right upper limb now moved about, exploring the bedclothes and the side of the bed. From this state she gradually recovered, and by December 26 she was able to get up. After a period of physical exercise she always became more alert, smiling and spontaneous in actions and speech, but she was still slower than normal. Except under very strong stimulus her emotional display was below average. She was still on occasions incontinent. At this time hypersomnia became evident. When left to herself she would often fall asleep, sometimes even while standing up. This tendency to sleep by day was only slightly relieved by benzedrine. On discharge from hospital on January 8, 1941, she was still pathologically drowsy, and she showed a hypopituitary type of habitus, primary optic atrophy with normal visual fields and acuity, fanning of the toes on stimulation of each sole, but no other neurological signs. Her basal metabolic rate was −10 per cent.

Throughout the whole nine months of observation her temperature fluctuated between 95° and 98·4°, but was usually about 97°. Her pulse showed no gross irregularities; at first its rate was 80, but subsequently it varied between 54 and 110, being slowest when the decorticate state and loss of consciousness were most profound. The fluctuations in temperature and pulse-rate did not entirely disappear after aspiration of the cyst and improvement in the general clinical state. At the height of coma and extensor rigidity the respirations were slowed to 10 and were deep and stertorous. There were never any sudden changes of colour, and no sweating.

During the nine months of observation she did not grow or change in weight. Her blood-pressure fell during the period from 108/70 to 75/45. She never menstruated or showed any secondary sexual characteristics, and she never had polyuria or polydipsia. Her blood sugar three hours after her midday meal was 0·130 per cent., and intravenous glucose produced no alleviation of the akinetic mutism.

When she was last seen in July, 1941, eight months after operation, she was well and free from headache, mutism, drowsiness and polyuria, and she was able to do routine work. She was forgetful, and still at times incontinent of urine. She still had not menstruated.

## Discussion.

The feature of this case which we wish to emphasize is the recurring episodes of mutism and immobility associated with pressure of a cyst of Rathke's pouch upon structures surrounding the 3rd ventricle.

The state was described by the child's parents and by nurses as one of drowsiness, but drowsiness was not a feature of it. Except during periods of severe decorticate rigidity, the girl was always easy to rouse and she did not sleep excessively. The most profound manifestations were mutism, loss of feeling tone, loss of emotional expression, of spontaneous and of most other voluntary movements, and total incontinence of urine and fæces of a degree and persistence rarely met with in brain lesions not associated with coma. She was incapable of originating active manifestations of any kind, with the notable exception that ocular fixation and movement occurred in response to the movement of external objects and to sounds. It was this which gave the child an appearance of alertness so incongruous with her silence and general immobility.[1]

All feeling appeared to be in abeyance. To painful stimuli there was reflex withdrawal and at times, after a prolonged latent period, a slow and ineffective voluntary movement of her hands towards the point of stimulation, but never any sign or sound of displeasure. The sight of food evoked no sign of pleasure, though at times she appeared to understand what it was and would make a slow, feeble attempt to put it in her mouth. Pleasant and unpleasant substances, hard foodstuffs that should at first have been chewed or sucked, were all swallowed promptly without any expression of pleasure, distaste, or discomfort. There was disturbance of consciousness at the highest levels, as evidenced by total amnesia for

[1] These ocular movements are comparable in some ways to the tendency to attend to every visual stimulus ("hypermetamorphosis") observed in macaques by Klüver and Bucy (1939) after bilateral removal of the temporal lobes, including the uncus and the greater part of the hippocampus; but our patient differed from the monkeys, in that her attention was attracted by movement of objects rather than by objects themselves, and no motor reaction followed movement and fixation of her eyes.

the period of the episode. Apart from the incontinence there was no disturbance of visceral activities, though examination of these was somewhat cursory.

This state was not always complete. At times she could understand speech, and then, after urgent and repeated commands, she would make slow and feeble attempts to carry out the requisite movement. There was also on one occasion recognition of a person by name. But it was at all times difficult to break through to a level of her understanding, and her eyes were easily drawn towards any moving object or sound.

Considering ætiological factors, the evidence shows that the akinetic mutism was not due to general rise of intracranial pressure. Sometimes intracranial pressure was in fact raised, on one occasion to over 600 mm. $H_2O$ in the lateral ventricle, but reduction of the pressure by withdrawal of cerebrospinal fluid was not followed by improvement in the akinetic mutism. Furthermore, on the three occasions immediately before the 3rd ventricle cyst was aspirated, when there was akinetic mutism, the pressures in the lateral ventricle were 190, 80 and 220 mm. $H_2O$ respectively.

With the peculiar state of akinetic mutism there were always associated some signs of bilateral pyramidal tract involvement. As the condition progressed the patient assumed a decorticate posture, and later she became comatose and had tonic fits. However, the loss of voluntary mobility and the mutism occurred before there was any increase of extensor tonus and assumption of the "decerebrate" attitude. Before the third aspiration of the cyst, when the mutism was developing and the characteristic electro-encephalographic findings were already present (q.v.), tone was normal in the limbs. It may be concluded that the state of akinetic mutism is not dependent on the presence of decorticate rigidity.

Aspiration of the 3rd ventricle cyst was followed by prompt return of vocalization, speech, interest in her surroundings, emotional feeling, and voluntary movement. The first time the cyst was emptied the child immediately emitted a grunt, the first sound she had made for at least a fortnight, and within ten minutes she moved and talked and asked where she was. During recovery there was no residual dysphasia, agnosia, or apraxia. At this stage it will be convenient to defer further consideration of the nature of this state until the electro-encephalographic findings have been reported.

*Electro-encephalographic findings.*—On two occasions we were able to follow by electro-encephalograms the course of clinical recovery after aspiration of the cyst. We used bipolar leads, R.C.C. amplifiers with an overall time-constant of one second,

and recorded with cathode ray tubes. The findings on the occasion of the second aspiration are set out in Table I, and illustrated in fig. 2.

TABLE I.—SECOND ASPIRATION OF CYST.

| *Date* | *Time* | *Clinical features* | *E.E.G.* |
|---|---|---|---|
| 18.10.40 | 2 p.m. | Akinetic mutism | 1. 8-waves of large amplitude from whole cortex, irregular, frequency about 2 per sec. Every ten seconds or so, bursts of markedly sinusoidal waves, of 200–300 micro-volts amplitude and frequency 2·5 per sec.; no localization |
| | 6.15 p.m. | As above | 2. As 1 above. Bursts somewhat more frequent, and of large amplitude (fig. 2*a*) |
| 19.10.40 | 4.30 a.m. | Tonic fit followed by coma | |
| | 11.45 a.m. | Cyst aspirated. 15 c.c. of brownish fluid obtained<br>Ventricular pressure 80 mm. $H_2O$ | |
| | 12 noon | Sat up spontaneously. Not responding verbally but ate a piece of biscuit | |
| | 12.15 p.m. | During e.e.g. says "Oh, dear" a number of times, but does not respond to questions | 3. As 2 above. No diminution of amplitude, or frequency of bursts |
| | 5.30 p.m. | Responding verbally, though still sluggish<br>Answers questions, names objects, cuts with scissors, obeys commands | 4. Diminution of amplitude; longer periods of quiescence between bursts |
| 20.10.40 | a.m. | Improved. More lively .. | 5. Bursts less frequent than in 4, shorter in duration, and of diminished amplitude. Between bursts, amplitude much diminished (fig. 2*b*) |
| 21.10.40 | p.m. | Further improved | 6. Small abnormality. Occasional vestiges of bursts seen (fig. 2*c*) |
| 24.10.40 | | Discharged. No neurological abnormality beyond bilateral primary optic atrophy | |

Similar findings were obtained on the third occasion of tapping the cyst, although the potential changes just previous to the operation were of smaller amplitude than on the previous occasion. The same general features were present—irregular slow discharge broken into by bursts of regular waves of greater amplitude which were synchronous in the two hemispheres (fig. 3*a*). This appearance was not affected by intravenous injection of 5 c.c. of 50 per cent. glucose. At this time the patient had not sunk into the completely developed akinetic mutism, although it was clear that this state was approaching, and there was no decorticate rigidity. She was drowsy and complaining of headache, and during the electro-encephalogram was reiterating "Oh, dear" in the stereotyped fashion previously noticed. The cyst was then aspirated for the third time, on November 18, 1940, and although clinical recovery was not so dramatic the electro-encephalogram carried out within half an hour of the operation already showed longer intervals between the bursts (fig. 3*b*). On the next day, when she was much improved and answering questions quite sensibly, electro-encephalography showed very slight abnormality, amounting to small, irre-

gular delta discharge (fig. 3*c*). Three weeks after aspiration of the cyst alpha-rhythm, which had been absent during the acute phases, was now present.

After operation the electro-encephalogram still showed large delta waves mainly from the occipital region, synchronized in the two hemispheres and without any bursts of sinusoidal waves. On discharge in January, 1941, her electro-encephalogram was normal except for occasional small delta waves (fig. 3*d*).

The characteristics of this picture were slow discharges of the order of 200-300 microvolts from the whole cortex at a rate of 1 to 3 per second,

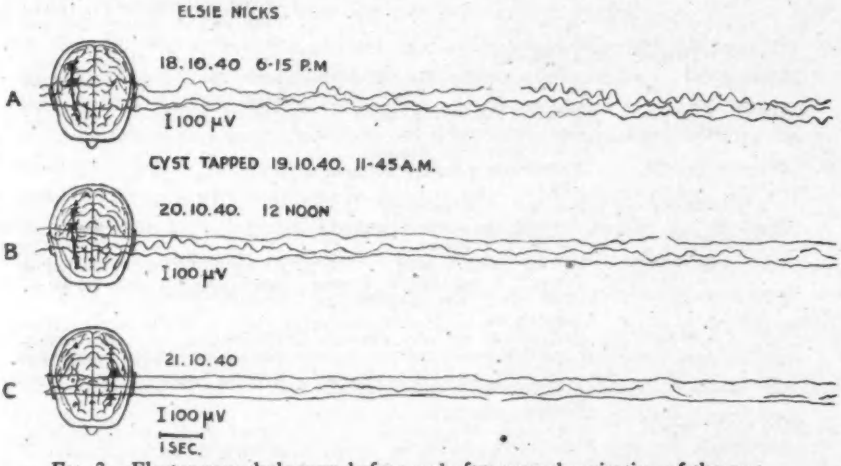


FIG. 2.—Electro-encephalogram before and after second aspiration of the cyst.

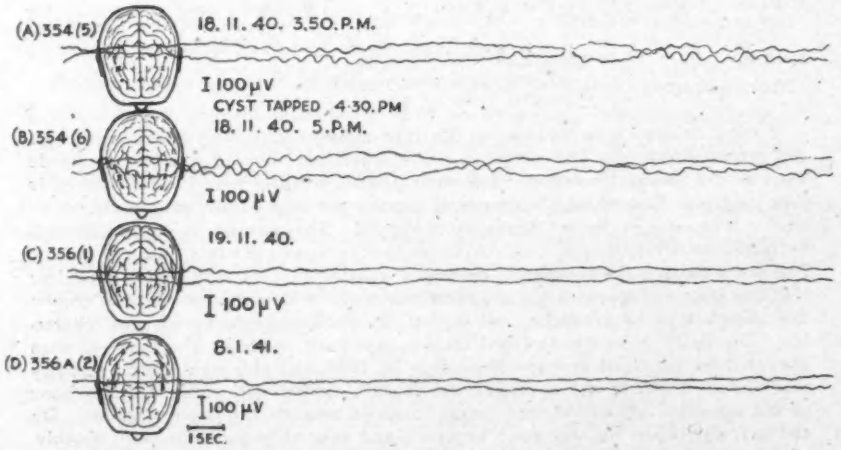


FIG. 3.—Electro-encephalogram before and after third aspiration of the cyst.

irregular in form and interspersed by periods of nearly sinusoidal discharge of slightly greater amplitude at 2 to 5 per second (figs. 2 and 3). These sinusoidal bursts have never been seen by us in states of raised intracranial pressure, and indeed there was no significant rise of intracranial pressure in our patient at the times of electro-encephalographic observation. Following aspiration of the cyst the abnormal wave form progressively disappeared with concomitant improvement and disappearance of mutism and akinesia.

Walter, Griffiths and Nevin (1939) have reported abnormalities of the electro-encephalogram in one case of tumour involving the structures around the 3rd ventricle, and causing a state of pathological sleep. They describe their patient's condition as " extremely drowsy," with deep, regular respiration at 20 per minute. She resisted any attempt to open her eyes, but " with further stimulation she could generally be roused, so that she would open her eyes, stir, perhaps smile, and mutter a few meaningless words. At other times she was more awake, and then it was noticed that she moved all her limbs normally and also opened her eyes on command." The patient was doubly incontinent, had no cranial nerve signs so far as it was possible to determine, and no papillœdema. Tone was normal in all limbs, reflexes were normal in the arms, but the abdominal and deep leg reflexes were absent. The plantars were flexor. At lumbar puncture the initial pressure of cerebrospinal fluid was 140 mm. After a few days the patient died in hyperthermia. At post-mortem examination a soft, greyish-red tumour, described as a perithelial sarcoma, was found, involving the thalamus, hypothalamus, internal capsule and corpus striatum on the left side, sparing only the posterior third of these structures. A wedge-shaped mass of tumour extended across the middle line towards an area of softening in the right globus pallidus. The infundibular stalk and pituitary were not involved.

The electro-encephalogram in this case showed continuous high-voltage slow discharge from all areas of the cortex. The potential changes reached a level of 500 microvolts. They were irregular in form, and poorly synchronized in the two hemispheres. " The discharge persisted almost unchanged when the patient was partially aroused. There was no trace of the normal alpha and beta rhythm, but the potential of the slow discharge decreased by about 20 per cent. when the patient was awakened as much as possible, the rhythm being the same."

The authors consider that such an electro-encephalographic picture more closely resembles that found in normal sleep than in the coma associated with raised intracranial pressure, in which, they state, " a very regular rhythm of smaller amplitude may be found in all areas." They

suggest that this difference "may be useful in differentiating between the stupor of hydrocephalus and that of hypersomnia associated with a local lesion in the hypothalamic region." It is quite debatable whether hydrocephalus disturbs consciousness except through the brain-stem. Certainly there are many cases of hydrocephalus and raised intracranial pressure in which there is no stupor or other disturbance of consciousness, and it may be that this is an attempt to differentiate by electro-encephalography between two states that are not really distinguishable. The important point is that in Walter, Griffiths and Nevin's case, as in ours, the electro-encephalographic picture was clearly associated with a lesion of the diencephalon rather than with a state of raised intracranial pressure. In our case the slow, irregular discharge, such as Walter, Griffiths and Nevin saw, is broken into by bursts of regular sinusoidal waves of 100-200 microvolts. The state of coma in Walter's case differed from the condition obtaining in our case, being more definitely like sleep, and the e.e.g. in their case more closely resembled that of normal sleep as described by Davis *et al.* (1938).

*The impairment of cortical function.*—In this clinical state of akinetic mutism the functions disturbed for the most part involve the cortex: voluntary movement, including speech, spontaneous activity of all kinds, emotional expression, perception and memory. There was as a rule absence of all forms of voluntary movement, except that of movement and fixation of the eyes in response to sound or a movement in the outside world, functions which in man, according to Holmes (1938), can be evoked only through the cortex.

There was no coarse lesion of the cortex or interruption of afferent cortical pathways from limbs or special sense organs in the ordinary way, no persisting motor or sensory palsy, beyond a fleeting left hemiparesis on one occasion which only served to show the essential contrast between this general somatic akinesia and a specific defect of motor pathways.[1] Furthermore, recovery from akinesia was quite unlike recovery from a cortical injury. There was no stage of weak or clumsy movement, no dysphasia or dyspraxia. Ten minutes after 3rd ventricle cyst was emptied the child sat up in bed and said: "Where am I?" It was more like waking from sleep.

During the height of the akinesia such cortical activity as was present was restricted, incomplete—a slow, deliberate movement of the hands to

[1] This temporary left hemiparesis after simple tapping of the right lateral ventricle may also indicate that the cortex in akinetic mutism, though healthy, is nevertheless abnormally vulnerable.

remove a strong nocuous stimulus of her trunk, but no sound, grimace, or tears, no movement of head or eyes towards the stimulated area; movement of the eyes towards a person coming within her field of vision, ocular fixation and even on one occasion recognition, but no smile of greeting, no speech; on one occasion a feeble and unsuccessful attempt to put a chocolate from her hand to her mouth, followed by chewing movements, but no attempt to look for the fallen chocolate.

Collateral evidence of the restriction of cortical activity is available from the electro-corticographic records of cats. Bremer (1937) has shown that when a sleeping animal is stimulated by a loud sound there is increased electrical activity confined to the auditory cortex. If the sound wakes the animal the whole cortex shows increased electrical activity. This rapid change from restricted to general cortical activity may be compared with the rapid recovery of our patient, when her cyst was tapped, from akinetic mutism to consciousness.

In cats, by section between the medulla oblongata and spinal cord without division of the blood-vessels, Bremer (1936*a*) has produced a state comparable clinically, though not electrically, with akinetic mutism. The preparation (encéphale isolé), which is kept alive by artificial respiration, is characterized by absolute immobility of the trunk and limbs in contrast with movements of the eyes and ears. The eyes follow the movements of objects in their field of vision and there are conjugate movements of eyes and ears in the direction of a noise; the pupils dilate on attention, but not with emotional stimuli. In spite of the existence of good buccal and masticatory reflexes, there is no facial expression of fear or anger. The cat appears to be feebly interested in its surroundings. From time to time it sleeps, but can be wakened easily by any stimulus that can still reach the brain.[1]

There is thus close similarity in behaviour between Bremer's preparation and our patient. Both respond to visual and auditory stimuli by eye movements, and, in the case of the cat, by movements of the external ears. All other higher level activity is restricted. In the cat this is due to anatomical division of the long ascending and descending pathways, with elimination of many afferent impressions from the body and the outside

[1] The electro-corticograms (Bremer, 1936*b*) were different from what was seen in our case. During sleep they showed a well-marked ten-a-second rhythm in all parts of the cortex. In this connection, however, it must be noted that Bremer used small coupling condensers in the amplifier so that low frequencies would be severely attenuated.

world. Can the restricted cortical activity in our patient be explained on the same basis?

In our case there was no gross interruption of the long pathways. Exteroceptive sensibility was not entirely lost and there was no paralysis in the commonly accepted sense of the term. If it had been possible to make the observation, electrical stimulation of the motor cortex during akinetic mutism might have been expected to produce the appropriate movements. If we are to regard this syndrome as due to pressure on the long motor and sensory pathways to and from the cortex then we must indeed envisage a selective variety of motor and sensory loss such as has not been encountered in other lesions of the long tracts. This explanation of the syndrome does not impress us as a satisfactory one, the more so as the impairment of function was affected at the diencephalic level by a cyst distending the 3rd ventricle. It is true that pressure of the cyst gave rise from time to time to decorticate rigidity, presumably from pressure on the extra-pyramidal pathways, but there is clear evidence, on at least one occasion, that the state of akinetic mutism preceded the onset of extensor hypertonus.

We suggest that the primary disturbance produced by the cyst was in the hypothalamic-thalamic pathways alongside the 3rd ventricle, and that the peculiar, readily reversible disturbance of the cortex was due to absence of a normal stream of impulses to the cortex from this source.

Electro-encephalographic studies in this case also show the disturbance of cortical activity that proceeds simultaneously with the clinical state, developing with it, regressing as it recedes (figs. 2 and 3). The sinusoidal burst seen in this case do not appear to have been recorded in other human cases; nor have they been seen in animals after complete deafferentation, though in this connection it must be noted that Bremer has used a recording technique which eliminates slow waves. It is difficult to offer an explanation of them, but Professor Adrian has suggested to us that, when pressed on by the cyst, the thalamus may have sent up abnormal volleys of impulses which were responsible for the episodes of large sinusoidal waves in the cortex.

*Akinetic mutism with other lesions.*—We have encountered incomplete varieties of akinetic mutism after ventriculography in strictures of the aqueduct of Sylvius and other mid-brain lesions. These cases do not present clear evidence as to the level of the lesion producing akinetic mutism and consequently they are not considered in detail here. The literature also is not very helpful. After encephalitis lethargica there sometimes supervenes a state which resembles akinetic mutism. In this

condition, according to v. Economo (1931, p. 125), there is akinésis without increase of muscular tone, and there is lack of emotional expression, sometimes with and sometimes without loss of feeling. In our case all emotional expression was lost and, so far as we could judge from the motor loss and loss of speech and amnesia, there was also loss of emotional feeling. Interpretation, however, must needs go cautiously, since according to Economo (1931, p. 162) " the attempt to deduce apathy from lack of mimic expression must be regarded as a failure."

Traut (1935) and Richter and Traut (1940) have reported a case of chronic encephalitis which in some respects evidently resembled our case. A young woman of 26 was under observation for over five years and during that time she did not speak, though she learned to grunt and evidently she understood. She was totally incontinent of urine and fæces. At times she would open her eyes and look at visitors, and she was easily roused from sleep. Though she was for a time inert she could move, resist, and smile, and she exhibited athetoid movements of the limbs. In other respects also she was unlike our patient. At necropsy typical lesions of chronic epidemic encephalitis were found in " the periventricular grey matter of the 3rd ventricle, the peri-aqueductal grey matter, the tegmentum of the mid-brain and pons, the globus pallidus, the anterior grey matter of the cord and, to a slighter degree, the corpus striatum and the thalamus." The outstanding lesion was in the posterior half of the hypothalamus: " the posterior hypothalamic nuclei were not found, and the nuclei of the mammillary bodies had completely disappeared." The cerebral cortex and cerebellum were " little affected ": in the cerebral cortex " mild to moderate non-specific changes in scattered ganglion cells and occasional small areas of cellular clearing were present, together with some diffuse increase of glia." Commenting on the clinical features the authors write: " The state was not coma in the ordinary sense of the term. It resembled precisely the peculiar trance-like condition which neurosurgeons not infrequently encounter in patients after operations in the neighbourhood of the 3rd ventricle and mid-brain."

*Anatomical considerations.*—It was shown in our case that the akinetic mutism did not depend on general rise of intracranial pressure; and the cortex itself was not affected by any coarse lesion, if we may judge from the rapid recovery of the clinical state and of the electro-encephalographic picture after aspiration of the 3rd ventricle cyst. In these circumstances we conclude that the symptoms were the direct result of pressure of the cyst upon the diencephalon. The 3rd ventricle was distended by the cyst (fig. 1) to such an extent that its lateral walls were stretched apart some

1·7 cm., as compared with the normal width of 0·15 to 0·3 cm. This degree of stretching of the 3rd ventricle is not exceptional in cases of cystic or solid epidermoid (Rathke pouch) tumours, in most of which, it must be noted, the syndrome of akinetic mutism is not seen. We have so far failed to find in the literature any case of 3rd ventricle cyst which could be definitely labelled as akinetic mutism, notwithstanding the remark of Richter and Traut quoted above. The symptoms most commonly seen with epidermoid tumour in the 3rd ventricle are headache and vomiting, depression or absence of sexual function, diabetes insipidus, adiposity, disturbance of temperature regulation, failure of vision, hypersomnia, and coma. The last two of these symptoms, and possibly also visual loss, might effectively mask akinetic mutism, but the capricious clinical effects of 3rd ventricle tumours cannot often be explained by supposing the dominance of one symptom over another.

While it is not possible to determine which particular structures in the wall of the 3rd ventricle were so compressed as to produce this syndrome, the absence of chiasmal signs suggests that the cyst did not encroach upon the hypophysis and its stalk. The optic tracts are shielded from pressure within the 3rd ventricle by the thalamus, and this doubtless contributes to the preservation of the eye movements that was such a noticeable feature of the clinical state. The presence of pupillary and external ocular movements excludes the pineal body, quadrigeminal plate, and the aqueduct of Sylvius. There remain as the main structures in the immediate neighbourhood of the cyst the thalamus, hypothalamus, and fornix.

As to the fornix, we have on several occasions divided the fornix completely across above and in front of the foramina of Monro, in the process of removing 3rd ventricle tumours, without producing any disturbance of consciousness. This brings us to a consideration of the connections between the hypothalamus, thalamus and cortex. As Le Gros Clark (1936) has shown, there are at least two main pathways (fig. 4). One is from the mammillary bodies to the anterior nucleus of the thalamus by the mammillo-thalamic tract, or bundle of Vicq d'Azyr, and the anterior nucleus is related by direct thalamo-cortical radiations with the whole length of the gyrus cinguli. Another and perhaps more important connection is between the posterior hypothalamic nucleus, which has been called a controlling centre for the sympathetic system, and the medial nucleus of the thalamus, which is known to be connected with the frontal pole of the cerebral hemisphere.

On the nature of the afferent connections of the medial nucleus of the

thalamus Le Gros Clark (1936) says: "It has been shown that fibres run up alongside the 3rd ventricle (forming a part of the periventricular system of fibres) and terminate in the medial aspect of the nucleus. It seems probable, on anatomical grounds, that these fibres relay impulses which have their origin in the hypothalamus. If the hypothalamus is regarded as a region for the central representation of visceral activities and as an essential element in the integration of emotional reactions, then the medial nucleus may be regarded as a relay station through which the neural processes associated with these activities are ultimately projected on to a cortical level." In this system fibres between hypothalamus and thalamus

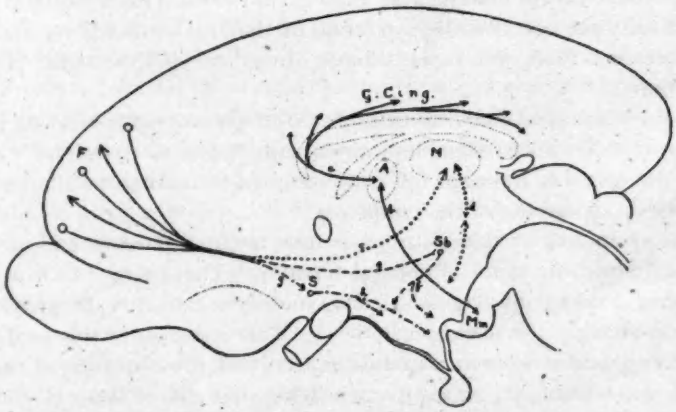


FIG. 4.—"Diagram showing the connections of the neopallial cortex with the hypothalamus. *A*. Anterior nucleus of the thalamus, connected to the gyrus cinguli (*G. Cing.*) by fibres (*a*). The anterior nucleus is connected to the medial mammillary nucleus by the bundle of Vicq d'Azyr (*b*). *F*. Frontal cortex connected with the medial nucleus of the thalamus (*M*), with the subthalamus (*Sb*), and with the septal area (*S*) by fibre paths (*c*, *e* and *g*). The medial nucleus is related to the posterior part of the hypothalamus by periventricular fibres (*d*). The subthalamus is connected to the hypothalamus by descending fibres (*f*), and the septal area is connected to the hypothalamus by a septo-hypothalamic tract (*h*)" (Le Gros Clark, 1936).

run immediately beneath the wall of the 3rd ventricle and might be particularly vulnerable to pressure when the 3rd ventricle is distended by cyst.

To regard akinetic mutism as due to involvement of these hypothalamic-thalamic connections is of course speculative. But the evidence of our case shows clearly that the cortex was largely put out of action by local pressure at the diencephalic level, and it suggests that normal cortical activity depends not only on afferent impulses from the outside world

and the somatic proprioceptors, but also on some other impulses mediated by the diencephalon. It may be, indeed, that consciousness depends on the synthesis of impressions from the outside world with those from the interior of the body, and that this is one of the main functions of the hypothalamic-thalamic system and its cortical connections.

## Summary.

A cyst distending the 3rd ventricle produced mutism, loss of voluntary and emotional movement, with the exception of movement of the eyeballs, apparent loss of emotional feeling, and other symptoms which are at present conveniently described as akinetic mutism. This syndrome was treated on three occasions by aspiration of the 3rd ventricle cyst and on each occasion there was rapid, almost immediate disappearance of the symptoms.

In the state of akinetic mutism electro-encephalography showed high voltage (200-300 microvolts) slow waves with bursts of sinusoidal waves. After the cyst was aspirated the e.e.g. returned to normal simultaneously with the disappearance of the symptoms.

The symptoms of akinetic mutism may be interpreted as due for the most part to disturbance of cortical function. They were shown not to be due to a rise of intracranial pressure and were evidently due therefore to disturbance at the diencephalic level. This disturbance was probably an interruption of afferent impulses, not only of those related to exteroceptive and somatic proprioceptive sensibility, but also of those of visceral sensibility.

We desire to thank Dr. Dorothy Russell for the pathological report and the Medical Research Council for providing a technical assistant for electro-encephalography.

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# A NOTE ON THE CLINICAL ANATOMY OF THE VEINS, WITH SPECIAL REFERENCE TO THE SPINAL VEINS.

BY H. A. HARRIS.

*(From the Department of Anatomy, University of Cambridge.)*

THE recent article on sagittal sinus thrombosis after childbirth by Dr. J. Purdon Martin (1941) and the references therein to the work of Dr. Oscar V. Batson (1941) of Philadelphia call for a brief statement on a few of the facts of clinical significance in the venous system of Man, and for a measure of caution in rediscovery of these well-established facts.

From the earliest days attention has been called to two types of veins, primary and secondary, which have developed from the primitive capillary network of early embryos. The primary veins are set away from the arteries, the secondary veins accompany the arteries, and so are often termed venæ comitantes. All these can be seen clearly in the early chick embryo—first an area vasculosa as a diffuse capillary network, secondly a peripheral venous sinus, and thirdly venæ comitantes accompanying the arteries which gradually emerge as main routes of supply in the primitive network of capillaries. This picture was familiar to Fabricus ab Aquapendente, the teacher of William Harvey.

In the dissecting room the student is apt to concentrate on the more recent type of vein, such as the axillary or femoral vein which runs with the accompanying artery. He does not realize that the cephalic and basilic veins in the upper limb and the saphenous and short saphenous veins in the lower limb belong to an older pattern. This is why he is so puzzled by the divorce of the cerebral arterial circle of Willis from the venous sinuses of the dura mater; the subintestinal vein of the embryo and the portal vein of the adult divorced from the mesenteric artery of the gut, and the hepatic vein from the hepatic artery; also in many forms the discrepancy in size and position between the arteries and veins of the neck, head and scalp, not to mention the arteries and veins of the dorsum of the hand and foot.

The arterial system of the young embryo presents a well marked ventral and dorsal aorta, with segmental arteries as the main feature, as in the branchial arteries and intercostal arteries. On the other hand, the

venous system of the embryo presents more numerous longitudinal venous trunks, as in the anterior, posterior and subcardinal veins, the pattern so familiar in the adult dogfish. In the arterial system certain longitudinal anastomoses joining together the segmental arteries appear, such as the internal mammary, superior epigastric and inferior epigastric, or the highly specialized vertebral artery of Man. In the venous system certain transverse anastomoses between the longitudinal venous trunks across the mid-line attract the attention, as the left innominate vein, between the anterior cardinal veins, the left renal vein crossing the aorta and the left common iliac bringing blood from the left hindquarter to the right-sided inferior vena cava.

If the processes of development are observed in the embryo, many rediscoveries of patent facts will be avoided. It is well known that the segmental (e.g. intercostal) artery, like the accompanying nerve and vein, divides into a posterior primary division for the skin and muscles of the back, and an anterior primary division for the anterior aspect of the trunk and the flank, including the limb bud.

The posterior primary division of the artery, passing posteriorly between the two ribs, gives off a spinal branch which enters the intervertebral foramen through which the spinal nerve issues, accompanied by a plexus of veins. This spinal branch divides into three branches; the posterior is termed the prelaminar branch, in front of the bony laminæ of the vertebræ; the anterior is called the postcentral, behind the bony centrum of the vertebra to which it gives a considerable supply; and the central branch, essentially neural, divides into anterior and posterior neural branches for the supply of the meninges and spinal cord. This picture of the segmental spinal arteries is modified by the appearance of longitudinal anastomoses, the anterior and posterior spinal arteries of adult anatomy, which arise from the vertebral, so that the primary segmental character is slightly obscured. The segmental spinal arteries persist in varying stages of development and there is one accompanying each spinal nerve as it issues from its intervertebral foramen. In the dorsal region they diminish in size, but there are frequently one or two of considerable size accompanying the 9th or 10th thoracic nerve.

On the anterior aspect of the spinal cord, at the level of the 10th or 11th pair of nerves, one of the segmental arteries becomes enlarged at the expense of its neighbours. This is the "great anterior spinal artery" of Adamkiewicz which sometimes supplies about 4 in. of the cord. This artery is frequently larger on the left side than the right. It may be of considerable significance in fracture of the lower dorsal spine with or

without crushing of the spinal cord. The relative suppression in size of some of the spinal arteries and the dominance of others is due to the fact that the spinal cord and spinal column, of the same length in the third month of fœtal life, grow at different rates, so that the spine of the male adult is 28 in. and the spinal cord only 18 in. long.

In the neck, where intercostal segmental arteries are suppressed, the spinal arteries arise from the longitudinal anastomoses of the vertebral artery, and between the skull and axis the spinal artery accompanying the first cervical nerve is the horizontal portion of the adult vertebral artery. These spinal arteries are often ignored by experimental physiologists when dealing with the spinal cord. Many of their nervous phenomena may be essentially of vascular origin, due to interference with the segmental arterial supply to the spinal cord or thrombosis in the corresponding veins.

The veins of the spine and spinal cord in the adult differ in many respects from the arteries, but the essential difference, as in the embryo, is the domination of the longitudinal channels, a condition so familiar in the vena azygos major. Accompanying each spinal nerve and spinal artery there is a plexus of spinal veins, in accord with the general rule that a nerve, artery or vital structure passing through a ligamentous or bony canal tends to be packed around with a plexus of veins and lymphatics. This is seen in the fibro-muscular inguinal canal where the vas deferens and spermatic artery is packed with a pampiniform plexus of veins, or the ascending part of the vertebral artery, traversing the foramina of the cervical vertebra, with a well marked vertebral plexus of veins.

Most modern textbooks, such as Quain and Gray, figure these spinal veins and copy the figures published by Breschet in 1829. Cruveilhier (1835) published a most detailed account of the veins of the spine and spinal cord, but the most accurate description and delineation was published by A. C. Bock at Leipzig (1823). This is the "Darstellung der Venen," with twenty copper-plate illustrations in colour. For some reason or other this monumental work of Bock has been ignored. Neither Martin nor Batson mention it, but this is excusable since it is not quoted even in the recent monograph on the veins by Franklin (1937).

Bock gives an account of the veins which is accurate and fits in with the known embryological and clinical facts. He displays the azygos system of veins in front of the spine and the rich venous plexus on the posterior aspect of the spine, calling particular attention to the rich anastomoses of the veins in the posterior muscles of the neck, the vena profunda

cervicis, the vertebral veins and the communications with the various cranial sinuses by way of the foramen magnum.

Inside the bony spinal canal Bock describes the rich venous plexuses, with dominance of the longitudinal anastomoses, between the bony canal and the theca of the dura mater (fig. 1). Inside the dura mater he depicts the well marked longitudinal anastomoses corresponding to the anterior and posterior spinal arteries.

Bock points out that the sacral branches of the internal iliac vein communicate freely with the veins within the spinal canal through the anterior spinal foramina, and the posterior plexus on the back of the sacrum does the same through the posterior sacral foramina. All the communications with the cranial venous sinuses are given in detail, also the communication through the mastoid emissary foramen, the posterior condyloid foramen and the circellus venosus hypoglossi accompanying the hypoglossal nerve through the anterior condyloid foramen. He pictures the transverse anastomoses between the two longitudinal channels, and describes a circellus venosus which is situated on the posterior aspect of the body of each vertebra, receiving large tributaries by two or more foramina from the marrow cavity of the vertebra.

Previous to the classical work of Bock on the veins, Winslow (1732) and Willis (1664) in particular had given clear accounts of the spinal veins (fig. 2). Willis described the internal longitudinal anastomoses of the spine as "vertebral sinuses" and depicted their communitions with the occipital sinuses of the dura mater. He also described in detail the vertebral plexus of veins accompanying the vertebral artery in the cervical transverse processes and the communications through the posterior condyloid foramen with the lateral sinus of the dura mater.

In Todd and Bowman's "Cyclopædia of Anatomy and Physiology" (1836-59) there is a comprehensive account of the venous system by B. Geo. McDowel. He says:

> "The veins of the parietes of the thorax (azygos veins), and those of the spinal column (rachidian veins), are remarkable as serving to connect the branches of the superior with those of the inferior vena cava."

Todd, in the article on the meninges, gives Breschet's picture of the longitudinal spinal sinuses on the posterior aspect of the bodies of the vertebræ, and says:

> "They have been known since the time of Fallopius, and were described by Willis as the longitudinal spinal sinuses."

In short, the anatomical picture of the spine and spinal cord is that of organs which have developed in the midst of segmental venous rings,

external and internal, joined by well developed longitudinal anastomoses, internal and external. All embryological growth of important organs tends to be in a rich arterial and venous field, preceded by an extensive

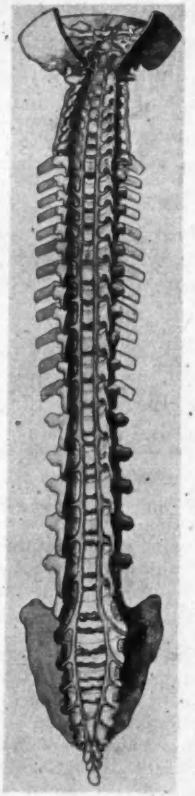

FIG. 1.

FIG. 2.

FIG. 1.—From Bock, "Darstellung der Venen," Leipzig, 1823, Tab. vii, fig. 1. The spine of a child seen from the dorsal aspect with the bony laminæ, spinal cord and meninges removed to show the venous sinuses (*sinus columnæ vertebralis*), the cross anastomoses (*circelli venosi*) and the communications with the cranial sinuses, vertebral plexus and spinal veins.

FIG. 2.—From Willis, "Cerebri Anatome," London, 1664, Tab. xiii, fig. iv. The internal longitudinal venous sinuses of the vertebral column with their numerous connections (*Sinus spinalis divaricationes in toto ductu ejus exhibet*). (The artist was probably Christopher Wren.)

capillary network, from which the adult pattern emerges. The vascular system is a great builder of main and secondary roads superimposed on primitive footpaths. By-passes and shunts are frequently devised as in the various transverse venous anastomoses, the ductus venosus, and many veins of the neck and limbs. The cephalic or "headward" vein of Man no longer runs to join the external jugular, but pierces the costo-coracoid membrane to join the axillary vein. The old route superficial to the mid-point of the clavicle is frequently the site of a well marked persisting cephalojugular vein, accompanying the middle descending supraclavicular nerve—often seen in the dilated superficial veins of the lactating woman.

*Venous Lakes and Backwaters.*

Batson is credited with suggesting that the spinal and vertebral veins are a storage lake as well as the pathway of drainage. Cloquet (1816) in Paris and Robert Knox (1831) in Edinburgh always described these veins as vertebral sinuses, and the only objection to the term sinus for these is that their relation to the dura mater differs from that of the cranial venous sinuses. John Hilton (1855) who, like John Hunter, receives so much lip service from anatomists, surgeons and physicians, was well aware of this system.

"The absence of valves in the whole of these venous tubes, is a circumstance which is doubtless connected with a wise intention. It enables the blood to pass in either direction, and consequently, greatly increases the freedom of venous circulation; a point of essential importance with an organ, whose functional capacity is so liable to interruption under so slight a disturbance of the balance of its circulating fluid."

Hilton also says, in his discussion on the numerous foramina for egress of veins:

"Children, whose actions are more influenced by sudden emotions, and less governed by the exercise of reason and judgment than adults, often exhibit prolonged fits of passionate crying; which, produce a considerable, though temporary excitement or increase of vascular action, and would often place them in imminent peril of their lives, were there not such efficient means provided for the rapid escape of blood from the interior of the cranial cavity under these sudden emergencies. Adults, on the other hand, having higher developed mental endowments, are more able to control their actions, and in some measure to check those fits of passion which lead to such increased vascular excitement of the cerebral organ."

From the time of Thomas Willis onwards the terms lacus, lacuna, laguna, lake and sinus appear repeatedly with reference to the veins of the skull, brain, spine and spinal cord. In 1853, Faivre described "les lacs sanguins," and they were rediscovered by Trollard in 1868. Later Tillaux and Labbé called them "lacs de sureté."

Charles Bell, even in 1823, had stated that the numerous vertebral and spinal veins and plexuses prevented the sudden and violent action of the muscles of respiration from injuring the smaller veins of the brain. Various pathologists from time to time called attention to the hæmorrhages in this system following urgent dyspnœa, myasthenia gravis and all cases of paralysis of the respiratory musculature.

Gowers (1888) was well aware of this venous system since he taught that 10 per cent. of all pyæmic metastases occurred in the brain and cranial venous sinuses.

### The Superior Longitudinal Sinus.

Martin refers to the internal structure of this sinus and says that in addition to the numerous trabeculæ, a continuous membrane may divide it horizontally for a portion of its length. He ascribes this partition to O'Connell.

Charles Bell (1823), in his textbook of Anatomy, describes the smaller bands as trabeculæ of Haller or chordæ of Willis, and states that "the sinus has in some rare instances been found of a square shape; its lower surface serving as a roof for another sinus of a triangular form, which for some way ran parallel with the great sinus, and which was of course also included in the lamina of the falx—these Malacarne calls *seni subalterni*"—i.e. subaltern or auxiliary sinuses.

### *Thrombosis of the Cerebral Sinuses.*

In 1861 the New Sydenham Society published a translation of the essay by von Dusch of Heidelberg on thrombosis of the cerebral sinuses. Out of a total of fifty-seven cases only two followed childbirth, and one case might be quoted as an example:

"*Case 43.*—A woman, aged 23, was twice attacked by peritonitis in the first week after delivery, for the cure of which repeated copious abstractions of blood were made during nine days. Twice, namely, forty leeches were applied, and twice twenty, or 120 altogether. A fortnight after delivery, headache and vomiting supervened, followed by hemiplegia. Great restlessness and screaming; coma, and finally, death three weeks after delivery. It must be added that, in consequence of these new symptoms, the patient was bled once from the arm, and fifteen leeches applied on various occasions.

*Sectio*—the superior longitudinal sinus was seen very much distended and of a glittering blackish appearance. It was filled by a clot, in the centre of which was a puriform fluid, resembling wine lees. The right lateral sinus, which was double at its commencement, also contained a similar fluid, blocked up in the direction of the jugular vein by a firm thrombus. The other sinuses quite normal. There were ecchymoses in the grey matter on the surface of the brain, especially in the course of the inflamed (thrombosed) veins on the convex surface and base. In the true pelvis, two deposits of pus; in the veins of the uterus, corresponding to the

place of attachment of the placenta, very firm, small, black plugs. In the cavity of the chest nothing abnormal."

Dusch was unaware of the role of the vertebral and spinal veins, for in his cases he says that a metastatic source of the thrombus in the sinus is "in the highest degree unreasonable, since any emboli detached thence would have to pass through two systems of capillaries (in the lung and brain) before reaching the venous sinus."

Notwithstanding that Virchow had already called attention to the fact that there are three sluggish venous areas in the body, the veins of the leg, the veins of the true pelvis, and the longitudinal and lateral sinuses of the brain, the neglect of well-established facts of anatomy produced the concepts of the paradoxical embolus and the thrombotic diathesis.

*Paradoxical Embolus.*

The absurd theories of paradoxical embolus still figure in textbooks of Pathology. They trace back to two cases published by Cohnheim (1889) and by Litten (1880), in which the foramen ovale was patent. Yet Cohnheim does state that "the locality to which the detached thrombi are transported is determined by anatomical conditions alone." The anatomical conditions were known to Willis, but the pathological implications were not recognized.

*Carcinoma and the Vertebral Veins.*

Breschet (1832) described the basiventral veins which issue from the marrow cavity of the vertebræ to join the circulus vasculosus between the spine and dura mater. They are figured in Gray and Quain. These veins leave the posterior aspect of the vertebræ by two large contiguous foramina. These foramina increase in size with age and may easily lead to errors in diagnosis in antero-posterior radiographs of the spine.

At University College Hospital, for the last twenty years, the teaching has been that in any carcinoma prone to give metastases in the bones, e.g. carcinoma of the thyroid, breast, testis, and prostate in particular, the 3rd lumbar vertebra should be searched on the radiograph for obvious signs of secondary growth. Even in cancer of the stomach such secondaries are often seen in the 3rd lumbar vertebra. According to the nature of the tumour and the tissue reaction of the host, the radiographic appearance and the histological section will show a wide range of abnormal appearances from local erosion to sclerosis.

*Cutaneous Veins.*

Batson illustrates the pattern of the subcutaneous veins by infra-red photographs. The subcutaneous plexus of veins is seen in the normal

person, more easily in both the well-trained athlete and the starveling child, in the lactating woman, and in any condition of muscular effort. These veins have been studied intensively by Thomas Lewis (1936) and his co-workers with special reference to the arterio-venous anastomoses and their function in rapid cooling of the venous blood brought to the surface from the deep muscular veins.

*Surgical Considerations.*

The block dissection of glands in the neck for carcinoma of the tongue involved wide interference with the veins of the neck. Wilfred Trotter repeatedly removed both the right and left sternomastoid muscles, the underlying internal jugular veins and the accompanying lymphatic glands. The patients displayed no subjective signs of cerebral congestion. There was marked post-operative œdema of the face, which gradually subsided, and at a later period bilateral pain over the insertion of the levator anguli scapulæ muscle into the superior angle of the scapula, presumably due to the fact that the weight of the limb was thrown on this muscle when the sternomastoid had been removed and the trapezius largely denervated. This extensive operation depended on the freedom of the venous anastomoses by way of the vertebral plexus accompanying the vertebral artery, the whole system of extra- and intra-spinal longitudinal anastomoses, the free anastomoses between veins of the scalp, diploë and the cranial venous sinuses. Particular attention to the role of the venous sinuses of the diploë as a backwater of the circulation was paid by Harris (1928, 1933).

Let us read Cruveilhier on general considerations of the veins of the spine:

"The veins of the spine are such that one may consider them, in relation to the general circulation, as establishing an uninterrupted communication between the veins of all parts of the trunk: so that one may conceive either of the two venæ cavæ to be obliterated, without interruption of the venous circulation. Even the vena azygos major, usually considered as the main route of communication between the superior and inferior vena cava, is not necessary, in view of the presence of the anterior and posterior rachidian plexus. This explains why I have seen at one time the inferior vena cava, at another the superior vena cava obliterated without any visible enlargement in the calibre of the azygos vein, and which may seem surprising, without any œdema either of the upper or lower limbs."

"If all the jugular veins were obliterated, the venous circulation of the head would persist nevertheless, and that by the venous circulation of the spine. In a dog I have ligatured both external jugular veins. The animal showed no sign of cerebral congestion; on exposure I found no enlargement of the small venules which accompany the carotid artery, venules which are rudimentary in the dog. Evidently, in this case, the circulation was maintained by the veins of the spine."

SUMMARY.

The cases of thrombotic and carcinomatous metastases, recently described by Batson and Martin, are explained in terms of long-established facts concerning the veins of the spine, spinal cord and meninges.

"For out of old fieldes, as men saithe
Cometh all this new corn fro yere to yere,
And out of old bookes, in good faithe
Cometh all this new science that men lere."

*Chaucer* (1328-1400), *The Assembly of Foules.*

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# INDEX

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## INDEX OF SUBJECTS.

### AUTHORS.


**Barrington, F. J. F.** PAGE

The Component Reflexes of Micturition ... ... ... ... ... 239

**Brain, W. Russell**

Visual Object-Agnosia with Special Reference to the Gestalt Theory 43

**Brain, W. Russell**

Visual Disorientation with Special Reference to Lesions of the Right Cerebral Hemisphere ... ... ... ... ... ... ... 244

**Cairns, H., Oldfield, R. C., Pennybacker, J. B., and Whitteridge, D.**

Akinetic Mutism with an Epidermoid Cyst of the 3rd Ventricle ... 273

**Denny-Brown, D., and Nevin, S.**

The Phenomenon of Myotonia ... ... ... ... ... ... 1

**Denny-Brown, D., and Russell, W. Ritchie**

Experimental Cerebral Concussion ... ... ... ... ... 93

**Denny-Brown, D., and Williams, D.**

Cerebral Electrical Changes in Experimental Concussion ... ... 223

**Dixon, G. J.**

Unilateral Exophthalmos (Proptosis): Causation and Differential Diagnosis ... ... ... ... ... ... ... ... ... 73

**Fitzgerald, G., and McArdle, B.**

Vitamins E and $B_6$ in the Treatment of Muscular Dystrophy and Motor Neurone Disease ... ... ... ... ... ... 19

**Hall, B., Noad, K. B., and Latham, O.**

Familial Cortical Cerebellar Atrophy ... ... ... ... ... 178

PAGE

**Harris, H. A.**
A Note on the Clinical Anatomy of the Veins, with Special Reference to the Spinal Veins ... ... ... ... ... ... ... 291

**Latham, O., Hall, B., and Noad, K. B.**
Familial Cortical Cerebellar Atrophy ... ... ... ... ... 178

**McArdle, B., and Fitzgerald, G.**
Vitamins E and $B_6$ in the Treatment of Muscular Dystrophy and Motor Neurone Disease ... ... ... ... ... ... 19

**Mitchell, D.**
Spinal Cord Degeneration produced by Dietary Means ... ... 165

**Nevin, S., and Denny-Brown, D.**
The Phenomenon of Myotonia ... ... ... ... ... ... 1

**Noad, K. B., Hall, B., and Latham, O.**
Familial Cortical Cerebellar Atrophy ... ... ... ... ... 178

**Oldfield, R. C., Cairns, H., Pennybacker, J. B., and Whitteridge, D.**
Akinetic Mutism with an Epidermoid Cyst of the 3rd Ventricle ... 273

**Pennybacker, J. B., Cairns, H., Oldfield, R. C. and Whitteridge, D.**
Akinetic Mutism with an Epidermoid Cyst of the 3rd Ventricle ... 273

**Riddoch, G.**
Phantom Limbs and Body Shape ... ... ... ... ... ... 197

**Russell, W. Ritchie, and Denny-Brown, D.**
Experimental Cerebral Concussion ... ... ... ... ... 1

**Smyth, G. E.**
The Significance of Lesions in the Dentate Nuclei Apparently Consecutive to Disease of the Frontal Lobes ... ... ... ... 63

**Whitteridge, D., Cairns, H., Pennybacker, J. B., and Oldfield, R. C.**
Akinetic Mutism with an Epidermoid Cyst of the 3rd Ventricle ... 273

**Williams, D., and Denny-Brown, D.**
Cerebral Electrical Changes in Experimental Concussion ... ... 223

## SUBJECTS.

PAGE

**Cerebellar atrophy, familial cortical.** Bruce Hall, K. B. Noad and O. Latham ... ... ... ... ... ... ... ... ... 178

**Concussion, experimental, cerebral.** D. Denny-Brown and W. Ritchie Russell ... ... ... ... ... ... ... ... ... 93

**Concussion, experimental, cerebral electrical changes in.** D. Williams and D. Denny-Brown ... ... ... ... ... ... ... ... 223

**Dentate nuclei, significance of lesions of, consecutive to disease of the frontal lobes.** G. E. Smyth ... ... ... ... ... ... 63

**Exophthalmos (proptosis), unilateral: causation and differential diagnosis.** G. Joly Dixon ... ... ... ... ... ... 73

**Micturition, the component reflexes of, Part III.** F. J. F. Barrington ... 239

PAGE

**Myotonia, the phenomena of.** D. Denny-Brown and S. Nevin ... ... 1

**Object-agnosia, visual, with special reference to the Gestalt theory.** W. Russell Brain ... ... ... ... ... ... ... 43

**Phantom limbs and body shape.** G. Riddoch ... ... ... ... ... 197

**Spinal cord degeneration produced by Dietary Means.** D. Mitchell ... 165

**3rd ventricle, epidermoid cyst of, with akinetic mutism.** H. Cairns, R. C. Oldfield, J. B. Pennybacker and D. Whitteridge ... ... 273

**Veins, a note on the clinical anatomy of, with special reference to the spinal veins.** H. A. Harris ... ... ... ... ... ... 291

**Visual disorientation with special reference to lesions of the right cerebral hemisphere.** W. Russell Brain ... ... ... ... ... 244

**Vitamins E and $B_6$ in the treatment of muscular dystrophy and motor neurone disease.** G. Fitzgerald and B. McArdle ... ... ... 19

# NOTICES OF RECENT PUBLICATIONS.


CEREBROSPINAL FEVER. PAGE

**Cerebrospinal Fever.** D. Brinton ... ... ... ... ... ... 90

EPILEPSY.

**Science and Seizures.** W. G. Lennox ... ... ... ... ... ... 195

INJURIES OF THE NERVOUS SYSTEM.

**Injuries of the Skull, Brain and Spinal Cord.** S. Brock ... ... ... 90

MEDICINE, CLINICAL.

**The Medical Aspect of Boxing.** E. Jokl ... ... ... ... ... 91

NEURO-OPHTHALMOLOGY.

**Neuro-Ophthalmology.** R. Lindsay Rea ... ... ... ... ... 91

NEURO-PSYCHIATRY.

**Foundations of Neuro-Psychiatry.** S. Cobb ... ... ... ... ... 195

NEUROSES.

**Lectures on War Neuroses.** T. A. Ross ... ... ... ... ... ... 196
**The Therapy of the Neuroses and Psychoses.** S. H. Kraines ... ... 196

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CONTENTS


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MAINWARING, JAMES. An Examination of the Value of the Empirical Approach to Aesthetics.

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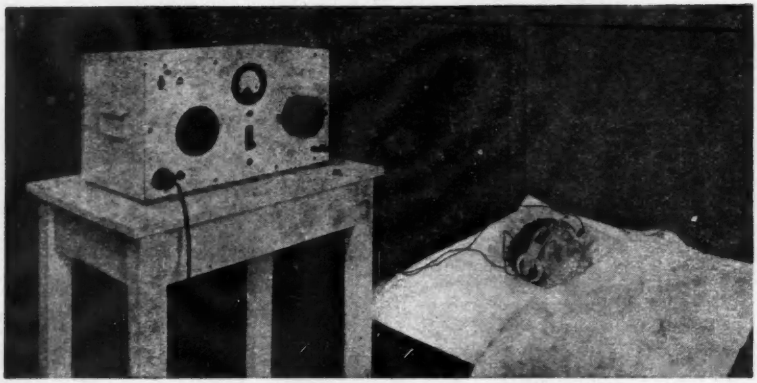

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